An elderly with repeated falls

IHGM
25/02/2014
To Cho Ting
Medical History

- Mr. Lam
- M/71
- NSND
- NKDA
- Lives with wife, son and daughter-in-law
- Daytime with wife
- Speaks Indonesian dialect and Cantonese
- Non vegetarian
- Education level: university
- Past medical history
  - HT  DM fu private for 3 years
  - Medications from private
    - Metformin sustained release 850 mg bd po
    - Glimepiride 2 mg daily po
    - Losartan 50 mg daily
1st admission on 15/12/2013 for fall while walking upstairs in the street
No LOC
No preceding symptoms
Mild head injury
No limbs injury
No epigastric pain/ tarry stool / coffee ground
Relatives reported patient had forgetfulness, mixing up pills, confused act at midnight and incontinence for a few months. no mood or behavioral problem

P/E
BP: 150/70, no postural drop
H’stic 5.8
Mild laceration over right forehead
CNS: GCS 14/15, 4 limbs power full, bilateral downgoing plantars, jerks normal, no focal neurological deficit
- Hb 11.4  MCV 78.2
  - Fe profile: Fe/TIBC 13.3/45.2  Fe sat 29%  ferritin 417
  - CEA 2.3
- Vit B12 99 pmol/L (133-675pmol/L)  folate 9 nmol/L
  - Anti parietal cell Ab –ve
  - anti intrinsic factor Ab –ve
- TSH normal, EIA TP –ve
- Chol 4.5 Tg 1.6 HDL 1.2 LDL 2.6
- CXR clear
- ECG SR RBBB
CT brain (plain):

- infarcts are noted in right thalamus, bilateral basal ganglia, corona radiata and capsular regions. Periventricular hypodensities are noted could be due to deep white matter ischemic changes due to small vessels disease.

- OGD showed fundal ulcer, no SRH, moderate antral erosion, biopsy: HP associated chronic gastritis
- Pantoloc 40 mg daily for gastric ulcer
- Vitamin B12 1000 mcg IMI started
- PT referred for walking exercise
  - Walks with one slight assistance
- OT referred for fall prevention intervention
- MMSE 15/30
- Hb remained static
- He was discharged
MMSE

- Orientation to time (1/5 points)
- Orientation to place (2/5 points)
- Registration of 3 words/objects (3/3 points)
- Attention and calculation (1/5 points)
- Recall of objects (1/3 points)
- Language (6/8 points)
- Visual construction (1/1 points)

MMSE Total: 15/30
• Second admission occurred one week after discharge
• Fall at home with abrasions over forehead and left knee
• No other sustained injury
• No LOC
• No preceding symptoms e.g. dizziness, palpitation or chest pain
- BP 140/80, no postural drop
- Neurological system
  - GCS 15/15
  - CN intact
  - Powers of 4 limbs: 4-/5
  - jerks normal
  - Bilateral downgoing plantar
  - Romberg sign +ve
  - Proprioception and vibration sense diminished
  - Light touch normal
- Chest, CVS, Abdomen unremarkable
- Blood tests unremarkable, Hb 12
• Blood glucose controlled with lower dose of metformin
• BP controlled with losartan and norvasc
• Aspirin was resumed with PPI
• Zocor was added
• Donepezil 5 mg was started

• PT assessment in acute ward
  • walk with frame and one assistance
• OT assessment
  • MMES 15/30
A course of training given in rehabilitation ward and GDH

- PT: walks unaided
- OT: BADL under supervision level
- MBI 64 → 82
- GDS: 1
- MMSE (two months later) 15/30
- Ophthalmologist assessment was normal
- Psychiatrist was referred
Problem List

- DM, HT, Hyperlipidemia
- Old CVA
- Dementia
- Recent GU
- Anaemia
- Vitamin B12 deficiency
- Repeated fall
Fall

Old CVA

GU

anemia

PPI

Vitamin B12 deficiency

Dementia

metformin

HT

Lipid

DM
Discussion

- Vitamin B₁₂ in elderly
  - metformin induced vitamin B₁₂ deficiency
- Dementia
  - vascular dementia
- Fall in dementia patient
Vitamin B12 deficiency

- What is the cause of vitamin B12 deficiency of this gentleman?
  - Intake insufficiency? Probably not
  - Food cobalamin deficiency? May be
  - Metformin?
Vitamin B12 deficiency in Elderly

- Occurs frequently among elderly patients
- Prevalence of around 20% among elderly people living in the community
- Often unrecognized or not investigated because the clinical manifestations are subtle
Causes of vitamin B12 deficiency

- **Food-Cobalamin Malabsorption:**
  - atrophic gastritis (40% in elderly persons); chronic gastritis.
- **Autoimmune:**
  - pernicious anemia, Sjogren’s syndrome.
- **Surgical:**
  - post-gastrectomy syndrome, ileal resection.
- **Decreased intake or malnutrition:**
  - vegetarians; chronic alcoholism; elderly people.
- **Intestinal malabsorption:**
  - chronic pancreatitis (exocrine insufficiency), Crohn’s disease, Whipple’s disease, celiac disease, amyloidosis, scleroderma, intestinal lymphomas or tuberculosis, tapeworm infestation, bacterial overgrowth.
- **Drugs:**
  - metformin, antacids, H2-blockers, PPIs, colchicine, cholestyramine, anticonvulsants, antibiotics, nitrous oxide.
- **Genetic:** transcobalamin II deficiency
Food-cobalamin malabsorption

Antacids: PPI/H2RA

Decreased intake in elderly

Metformin inhibits cobalamin-IF complex absorption
Major clinical manifestations of cobalamin deficiency

<table>
<thead>
<tr>
<th>System</th>
<th>Manifestation</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematological</td>
<td>Macrocytosis; hypersegmentation of the neutrophils; regenerative macrocytic anemia; medullary megaloblastosis (“blue spinal cord”)</td>
<td>Frequent</td>
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<tr>
<td></td>
<td>Isolated thrombocytopenia and neutropenia; pancytopenia</td>
<td>Rare</td>
</tr>
<tr>
<td></td>
<td>Hemolytic anemia; thrombotic microangiopathy (presence of schistocytes)</td>
<td>Very rare</td>
</tr>
<tr>
<td>Neuropsychiatric</td>
<td>Combined sclerosis of the spinal cord</td>
<td>Classic</td>
</tr>
<tr>
<td></td>
<td>Polyneurites (especially sensitive ones); ataxia; Babinski’s phenomenon</td>
<td>Frequent</td>
</tr>
<tr>
<td></td>
<td>Cerebellar syndromes affecting the cranial nerves, including optic neuritis, optic atrophy, urinary or fecal incontinence</td>
<td>Rare</td>
</tr>
<tr>
<td></td>
<td>Changes in the higher functions, even dementia, stroke and atherosclerosis (hyperhomocysteinemia); Parkinsonian syndromes; depression</td>
<td>Under study</td>
</tr>
<tr>
<td>Digestive</td>
<td>Hunter’s glossitis; jaundice; lactate dehydrogenase and bilirubin elevation (“intraduillary destruction”)</td>
<td>Classic</td>
</tr>
<tr>
<td></td>
<td>Resistant and recurring mucocutaneous ulcers</td>
<td>Rare</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain; dyspepsia; nausea; vomiting; diarrhea; disturbances in intestinal functioning</td>
<td>Debatable</td>
</tr>
<tr>
<td>Gynecological</td>
<td>Atrophy of the vaginal mucosa and chronic vaginal and urinary infections (especially mycosis); hypofertility and repeated miscarriages</td>
<td>Under study</td>
</tr>
<tr>
<td>Other</td>
<td>Venous thromboembolic disease; angina (hyperhomocysteinemia)</td>
<td>Under study</td>
</tr>
</tbody>
</table>
Neuropsychiatric symptoms due to vitamin B12 deficiency

- Often precede hematologic abnormalities

- Include motor, sensory, and autonomic symptoms; subacute combined degeneration of cord, cognitive impairment; mood and psychotic symptoms.

- Incidence 4%–50%

- Symptoms include paresthesia, ataxia, proprioception and vibration loss

- Memory loss, delirium, dementia

- Depression, mania, hallucinations, delusions, personality change, and abnormal behavior.
• Proposed mechanism
  • accumulation of homocysteine, which is linked to cognitive declined
  • accumulation methylmalonic acid, which is neurotoxic
  • demyelination of white matter

• Symptoms may not be reversed with vitamin B12 supplement.
  • The cognitive function of mild to moderate demented older people with vitamin B12 deficiency did not significantly change with vitamin B12 supplementation over a 10-month period. But the supplementation might have reduced the degree of delirium associated with dementia in one study

Vitamin B12 deficiency caused by drugs

- Proton pump inhibitor / H2 receptor antagonist
- PPI irreversibly inhibit the proton pump to prevent acid secretion into the gastric lumen

- H2RAs decrease acid secretion by the gastric parietal cells

- Protein bound cobalamin may not be adequately released from food in the presence of achlorhydria secondary to these therapy
Vitamin B12 deficiency caused by drugs

- **Metformin**
- Chronic metformin use results in vitamin B12 deficiency in 30% of patients
- Metformin disrupts the ileal vitamin B$_{12}$ absorption.
  - The vitamin B$_{12}$-intrinsic factor complex is dependent on the luminal calcium concentration to facilitate uptake by the ileal cell surface receptor
  - whereas metformin is believed to give a positive charge to the surface of the membrane, which would act to displace divalent cations such as calcium.
  - Impaired calcium availability due to metformin activity would therefore interfere with the calcium-dependent process of vitamin B$_{12}$ absorption
- Dietary calcium supplementation reverses metformin-induced vitamin B$_{12}$ malabsorption.

Bauman WA et al Diabetes Care 23:1227–1231, 2000
Risk Factors of Vitamin B$_{12}$ Deficiency in Patients Receiving Metformin

Rose Zhao-Wei Ting, MBBS; Cheuk Chun Szeto, MD, FRCP; Michael Ho-Ming Chan, FRCPA, FHKCPath; Kwok Kuen Ma, MBBS; Kai Ming Chow, MRCP

**Background:** Identification of risk factors for metformin-related vitamin B$_{12}$ deficiency has major potential implications regarding the management of diabetes mellitus.

**Methods:** We conducted a nested case-control study from a database in which the source population consisted of subjects who had levels of both serum vitamin B$_{12}$ and hemoglobin A$_{1c}$ checked in a central laboratory. We identified 155 cases of diabetes mellitus and vitamin B$_{12}$ deficiency secondary to metformin treatment. Another 310 controls were selected from the cohort who did not have vitamin B$_{12}$ deficiency while taking metformin.

**Results:** A total of 155 patients with metformin-related vitamin B$_{12}$ deficiency (mean ± SD serum vitamin B$_{12}$ concentration, 148.6±40.4 pg/mL [110 ± 30 pmol/L]) were compared with 310 matched controls (466.1±330.4 pg/mL [344 ± 244 pmol/L]). After adjusting for confounders, we found clinically important and statistically significant association of vitamin B$_{12}$ deficiency with dose and duration of metformin use. Each 1-g/d metformin dose increment conferred an odds ratio of 2.88 (95% confidence interval, 2.15-3.87) for developing vitamin B$_{12}$ deficiency ($P<.001$). Among those using metformin for 3 years or more, the adjusted odds ratio was 2.39 (95% confidence interval, 1.46-3.91) ($P=.001$) compared with those receiving metformin for less than 3 years. After exclusion of 113 subjects with borderline vitamin B$_{12}$ concentration, dose of metformin remained the strongest independent predictor of vitamin B$_{12}$ deficiency.

**Conclusions:** Our results indicate an increased risk of vitamin B$_{12}$ deficiency associated with current dose and duration of metformin use despite adjustment for many potential confounders. The risk factors identified have implications for planning screening or prevention strategies in metformin-treated patients.

*Arch Intern Med.* 2006;166:1975-1979
• A nested case-control study
• 155 patients with metformin-related vitamin B₁₂ deficiency
• 310 matched control
• Showed statistical significant association of vitamin B₁₂ deficiency with dose and duration of metformin use
• Among those using metformin for 3 years or more, the adjusted odds ratio was 2.39 (CI 1.46- 3.91)
Two case reports from CUHK in 2006 showed that macrocytic anaemia, neuropathy and cognitive impairment were caused by metformin induced vitamin B12 deficiency.

In both cases symptoms can be reversed after vitamin B12 replacement.
Long term treatment with metformin in patients with type 2 diabetes and risk of vitamin B-12 deficiency: randomised placebo controlled trial

Jolien de Jager, resident,1 Adriaan Kooy, internist,2 Philippe Lehert, professor of statistics,3 Michiel G Wulffela, general practitioner,3 Jan van der Kolk, biochemical engineer,3 Daniël Bets, program manager,3 Joop Verburg, chief laboratory attendant,3 Ab J M Donker, professor of internal medicine,7 Coen D A Stehouwer, professor and chair8

Vitamin B-12

<table>
<thead>
<tr>
<th>Concentration (pmol/L)</th>
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<tbody>
<tr>
<td>400</td>
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<tr>
<td>370</td>
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<tr>
<td>340</td>
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<tr>
<td>310</td>
</tr>
<tr>
<td>280</td>
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<tr>
<td>250</td>
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<table>
<thead>
<tr>
<th>Metformin group</th>
<th>Placebo group</th>
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</thead>
<tbody>
<tr>
<td>192</td>
<td>153</td>
</tr>
<tr>
<td>173</td>
<td>140</td>
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<tr>
<td>153</td>
<td>122</td>
</tr>
<tr>
<td>140</td>
<td>124</td>
</tr>
</tbody>
</table>

Number of available samples

Metformin group

Placebo group

Progressive decrease in vitamin B-12 levels in patients on metformin in 52 months
Diabetic patients with Alzheimer disease (AD) (n = 480) or mild cognitive impairment and those who were cognitively intact (n = 687) were included. Worse cognitive performance was associated with metformin use.

Patients with diabetes who used calcium supplements were less likely to be cognitively impaired.
CONCLUSIONS — Metformin exposure may be an iatrogenic cause for exacerbation of peripheral neuropathy in patients with type 2 diabetes. Interval screening for Cbl deficiency and systemic Cbl therapy should be considered upon initiation of, as well as during, metformin therapy to detect potential secondary causes of worsening peripheral neuropathy.

*Diabetes Care* 33:156–161, 2010
A prospective case-control study

Patients with type 2 diabetes and concurrent symptomatic peripheral neuropathy

Comparing those who had received 6 months of metformin therapy (n=59) with those without metformin exposure (n=63).

Metformin-treated patients had depressed Cbl levels, more severe peripheral neuropathy in these patients were identified
Association of Biochemical B₁₂ Deficiency With Metformin Therapy and Vitamin B₁₂ Supplements


Lael Reinstatler, mph
Yan Ping Qi, mph
Rebecca S. Williamson, mph
Joshua V. Garn, ms
Godfrey P. Oakley Jr., MD, MSPM

OBJECTIVE—To describe the prevalence of biochemical B₁₂ deficiency in adults with type 2 diabetes taking metformin compared with those not taking metformin and those without diabetes, and explore whether this relationship is modified by vitamin B₁₂ supplements.

RESEARCH DESIGN AND METHODS—Analysis of data on U.S. adults ≥50 years of age with (n = 1,621) or without type 2 diabetes (n = 6,867) from the National Health and Nutrition Examination Survey (NHANES), 1999–2006. Type 2 diabetes was defined as clinical diagnosis after age 30 without initiation of insulin therapy within 1 year. Those with diabetes were classified according to their current metformin use. Biochemical B₁₂ deficiency was defined as serum B₁₂ concentrations ≤148 pmol/L and borderline deficiency was defined as >148 to ≤221 pmol/L.

RESULTS—Biochemical B₁₂ deficiency was present in 5.8% of those with diabetes using metformin compared with 2.4% of those not using metformin (P = 0.0026) and 3.3% of those without diabetes (P = 0.0002). Among those with diabetes, metformin use was associated with biochemical B₁₂ deficiency (adjusted odds ratio 2.92; 95% CI 1.26–6.78). Consumption of any supplement containing B₁₂ was not associated with a reduction in the prevalence of biochemical B₁₂ deficiency among those with diabetes, whereas consumption of any supplement containing B₁₂ was associated with a two-thirds reduction among those without diabetes.

CONCLUSIONS—Metformin therapy is associated with a higher prevalence of biochemical B₁₂ deficiency. The amount of B₁₂ recommended by the Institute of Medicine (IOM) (2.4 µg/day) and the amount available in general multivitamins (6 µg) may not be enough to correct this deficiency among those with diabetes.

Diabetes Care 35:327–333, 2012
• Adults ≥ 50 years of age with (n = 1,621) or without type 2 diabetes (n = 6,867)

• Among those with diabetes, metformin use was associated with biochemical B12 deficiency (adjusted odds ratio 2.92; 95% CI 1.26–6.78).

• Consumption of any supplement containing B12 was not associated with a reduction in the prevalence of biochemical B12 deficiency among those with diabetes

• The amount of B12 in general multivitamins (6 mcg) may not be enough to correct the deficiency among those with diabetes
Management

- Rule out concomitant conditions which may further increase risk for vitamin B12 deficiency
  - suboptimal dietary intake and malabsorption due to atrophic gastritis, pernicious anemia etc.

- Screening for vitamin B12 deficiency for all adult patients with diabetes, especially those taking metformin at intervals of 1-2 years.

- Consider vitamin B12 and calcium supplementation in patients receiving metformin, particularly for older patients and for patients who have received metformin for several years.
## Therapeutic management of cobalamin deficiency using cobalamin treatment

<table>
<thead>
<tr>
<th>Route of administration</th>
<th>Initial treatment</th>
<th>Maintenance treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parenteral *</td>
<td>1000 mcg/d for 1 week, then 1000 mcg/wk for 1 month</td>
<td>1000 mcg/month until the cause of deficiency is corrected or for life in case of pernicious anaemia</td>
</tr>
<tr>
<td>Oral #</td>
<td>1000 mcg/d for 1 month</td>
<td>125-500 mcg/d for intake deficiency and food cobalamin malabsorption; 1000 mcg/d for pernicious anaemia</td>
</tr>
</tbody>
</table>

*Regardless of the cause of vitamin B12 deficiency

# For intake deficiency, food cobalamin malabsorption and pernicious anaemia
What is the cause of dementia of this gentleman?

- Alzheimer’s disease
- Vascular dementia
- Vitamin B12 deficiency
- Mixed dementia
Dementia

- Dementia is characterized by a decline in intellectual function affecting memory and other cognitive functions which occurs in clear consciousness.

- Usually leading to impaired functioning of daily activities.

- Affects approximately 5–7% of people aged over 60 years.
Causes

- Neurodegenerative
  - Alzheimer’s disease
  - Lewy bodies dementia
  - Frontotemporal
- Vascular causes
  - Multi infarct dementia
  - Subdural hematoma
- Metabolic causes
  - Vitamin B12 deficiency
  - Thyroid disease
  - Alcohol-related
- Infectious causes
  - Neurosyphilis, HIV
- Structural causes
  - Brain tumor, normal pressure hydrocephalus
- Inflammatory causes
- Miscellaneous
- Mixed type
Diagnosis

- MMSE (Mini-mental state examination)
- Clock drawing test
- Formal neuropsychological test
  - for atypical presentations
• **Standard tests**
  - Non contrast CT brain
  - Metabolic panel
    - CBC, serum vit B12, thyroid profile, depression screening
• **Tests in selected circumstances**
  - MRI brain
    - More sensitive for microvascular changes in vascular dementia
  - EEG
    - Slowing in AD
  - CSF
    - Neurosyphilis
  - Cerebral PET
  - SPECT scan
    - Biparietal hypometabolism in early stage of AD
Vascular dementia

- Encompasses a variety of vascular-related causes of dementia, including multiinfarct dementia (MID) and small vessel disease.

- Approximately 20% of all dementia cases

- The second most common form of dementia.

- It is equally prevalent in males and females

- Risk factors are similar to those for cardiovascular illness (including diabetes, hypercholesterolemia, hyperhomocysteinemia, hypertension, cigarette smoking, and physical inactivity).
Vascular dementia: causes

- Recurrent or specifically localized embolic strokes (from sources such as the heart or carotid artery, or local thromboses of larger-caliber intracranial vessels)

- Smaller subcortical strokes (e.g., lacunar infarctions) in gray and white matter structures

- White matter disease without clearly symptomatic strokes or gross tissue damage may cause insidiously progressive cognitive decline (Binswanger’s disease)

- Vascular dementia due to hemorrhagic lesions
Variable clinical features that are dependent on the localization of the vascular lesions.

- Focal neurological signs and symptoms
- Left hemisphere lesions tend to cause language problems
- Right hemisphere lesions tend to cause visuospatial problems

- Embolic or large-vessel stroke-related dementia may progress in a characteristic stepwise pattern, with intervening periods of stability punctuated by abrupt declines in cognitive function

- Multiple small subcortical infarcts may cause a more insidious decline, even in the absence of recognized stroke symptoms.

- Presents with signs and symptoms of both cortical and subcortical dementia
  - E.g. inattention, poor concentration, poor motivation

- Behavioral and psychological symptoms
  - Depression, apathy, mania, anxiety, emotional lability, agitation, psychosis, delusion.
- The main difficulty in diagnosing vascular dementia is distinguishing it from AD.

- Classically, vascular dementia is distinguished from AD based on an abrupt onset and a stepwise course.

- Prominent executive dysfunction and preserved recognition memory are also suggestive of vascular dementia.

- However, in many cases the symptoms of vascular dementia overlap with those of AD.

- Autopsy studies show that the co-occurrence of AD and CNS vascular pathology is not infrequent; the interaction may cause cognitive impairment that might not otherwise occur if the same level of AD or vascular dementia pathology was present alone.
Vascular dementia: treatment

- Control of vascular risk factors (e.g., hypercholesterolemia, hypertension, inactivity, diabetes, excess alcohol use, cigarette smoking, and hyperhomocysteinemia).

- If strokes are found on brain imaging studies, a stroke work-up should be initiated to determine if surgery (e.g., for carotid stenosis), anticoagulation (for atrial fibrillation), or antiplatelet agents (e.g., for small-vessel strokes) are indicated.

- According to AHA/ASA scientific statement
  - Donepezil can be useful for cognitive enhancement
  - Galantamine can be beneficial for individuals with mixed AD/VD

- Treat neuropsychiatric features (e.g., depression or psychosis)
Fall

- What is the risk factors of repeated fall of this gentleman?
  - Old CVA?
  - Dementia?
  - Vitamin B12 deficiency?
  - Anaemia?
  - Multifactorial
Definition

- Inadvertently come to rest on ground or a lower level

- Most investigators exclude conditions associated with LOC (seizure), paralysis (stroke), or violent events (severe blow)

- Syncope is not excluded
• Approximately 30% of adults aged > 65 years fall at least once each year

• The prevalence increases to nearly 50% in those aged over 85 years

• One percent of falls by individuals aged 65 and over result in hip fracture

• Between 14 and 22% of community-dwelling older people suffer recurrent falls (≥2 falls)
Fall and cognitive impairment/dementia

- The incidence of falls with cognitive impairment and dementia is more than twice that of cognitively intact older people with an annual incidence of approximately 60%

- Cognitive impairment is an independent risk factor for fall

- The incidence of multiple falls is also doubled

- Injurious falls are also more prevalent in older adults with dementia, with more than a threefold increase in hip fracture and poorer outcomes including institutionalization and death
Risk factors of fall in cognitive impairment/ dementia

- Psychosocial & demographic factors
  - Advanced age
  - History of falls
  - Walks with an aid
  - Inactivity
  - ADL limitations
- Medical factors
  - Dementia duration and severity
  - Parkinson's disease/parkinsonism
  - Arthritis/musculoskeletal complaint
  - Symptomatic orthostatic hypotension
  - Peripheral neuropathy
  - Autonomic neuropathy
  - Cardiac arrhythmia
  - Cataracts
  - Impaired vision
- Medication factors
  - Psychoactive medications
  - Antidepressants
  - Cardiovascular medications
  - Polypharmacy

- Balance & mobility factors
  - Impaired stability when standing
  - Impaired gait and mobility

- Sensory & neuromuscular factors
  - Muscle weakness
  - Slow reaction time
Neuropsychological factors

- Wandering/behavioral factors
- Attention and orientation
- Poor memory
- Depression/depressive symptoms
- Impaired executive function
- Anxiety
Multifactorial fall risk assessment

- Obtain relevant medical history, P/E, cognitive and functional assessment
- Determine multifactorial fall risk
  - History of falls
  - Medications
  - Gait, balance and mobility
  - Visual acuity
  - Other neurological impairments
  - Muscle strength
  - Heart rate and rhythm
  - Postural hypotension
  - Feet and footwear
  - Environmental hazards
Assessments of falls (functional)

- Functional gait & balance
  - Timed Up & go test
  - Berg Balance scale
- Mobility
  - walking aids, personal assistance, range
- ADLs
  - bathing, dressing etc.
Multifactorial/multicomponent intervention

- Minimize medications (esp. psychoactive)
- Provide individually tailored exercise program
- Treat vision impairment (including cataract)
- Manage postural hypotension
- Manage heart rate and rhythm abnormalities
- Supplement vitamin D
- Manage foot and footwear problems
- Modify the home environment
- Provide education and information
# Medications and fall risk

## Fall risk increasing drugs
- Antihypertensive agents
- Diuretics
- B blockers
- Sedatives and hypnotics
- Neuroleptics and antipsychotics
- Antidepressants
- Benzodiazepines
- Narcotics
- NSAID

## Odd ratio
- 1.24 (95% CI, 1.01-1.50)
- 1.07 (95% CI, 1.01-1.14)
- 1.01 (95% CI, 0.86-1.17)
- 1.47 (95% CI, 1.35-1.62)
- 1.59 (95% CI, 1.37-1.83)
- 1.68 (95% CI, 1.47-1.91)
- 1.57 (95% CI, 1.43-1.72)
- 0.96 (95% CI, 0.78-1.18)
- 1.21 (95% CI, 1.01-1.44)

Meta-analysis of the impact of 9 Medication Classes on Falls in Elderly Persons Woolcott et al. [2009]

The use of sedatives and hypnotics, antidepressants, and benzodiazepines demonstrated a significant association with falls in elderly individuals.
Fall and dementia

- According to AGS/BGS clinical practice guideline on fall prevention

- There is insufficient evidence to recommend, for or against, single or multifactorial interventions in community-living older adults with known cognitive impairment

- In a systematic review of 11 RCTs of cognitively impaired subjects. The investigators observed only limited effectiveness of physical training or exercise in reducing fall risk

- A study of education as part of a multicomponent intervention program observed that education was associated with a significant intervention effect on falls in the group with higher Mini-Mental State Examination scores but not in the group with lower scores

- The effectiveness of a customized multicomponent intervention after multifactorial clinical assessment was investigated in older patients with cognitive impairment and dementia presenting to the emergency department after a fall.
  - No significant difference between the intervention and control groups in fall risk was found
• Thank you