

# [Alzheimer’s disease case study](https://assignbuster.com/alzheimers-disease-case-study/)

* B. Trimble

Case Study

M. T. an 86-year-old Asian male is brought into the geriatric clinic by his daughter because he is becoming more forgetful. The daughter explains that the patient often does not even recognize his own grandson. When asked, however, the patient denies memory impairment. The daughter states that her father has been having trouble for almost four years now. She said she did not realize how much her father had changed until she watched a home video of her father with his grandson from six years ago. “ His personality has even seemed to have changed,” said the daughter. M. T. is no longer able to take care of his house and household chores and is sometimes slow to respond to questions.

Past Medical History

Peptic Ulcer Disease (PUD) with the last occurrence three years ago.

Social History

Retired sanitation worker times fifteen years. Lives with his daughter since his wife died five years ago. Previous cigarette smoker quit fifteen years ago. Denies ethyl alcohol (ETOH), and intravenous drug abuse.

Family History

Father died in combat in Korea

Mother died at age 92, unknown cause

Medications and allergies

NKDA

Prevacid 30 mg orally once daily Mylanta 30 ml orally as needed for stomach upset

Aspirin EC 81 mg orally once daily Tylenol 325 mg orally as needed for headache

Physical Examination

General– patient is a thin, pleasant man, with working memory in no acute distress.

Vital signs– BP 145/78 , pulse 80 RR 17 , WT 70 kg , Ht 6’2” BMI 19. 8

HEENT- Pupil’sPERLA Ears with cerumenNeck –no bruit no JVD

Cardiovascular– Normal sinus rhythm, S1 S2, negative for S3/S4, resp RRR

Neuro– Aox1 (Oriented to person only), CN – XII – XII intact, reflex normal

Abdomen– positive bowel sounds, non-tender, non-distended

External– WNL, no clubbing, cyanosis, or edema

Laboratory Tests

Albumin –(3. 6-5 g/dL) 3. 6g/dLtotal protein– (6. 3-8. 2g/dL) 6. 8g/dL

Alk Phos– (38-126U/L) 41U/LALT –(7-58 U/L) 21 U/L

AST– (7-58 U/L) 21 U/Lbilirubin– (0. 2-1. 3ug/dL) 0. 3ug/dL

BUN –(8-25mg/dL) 11mg/dLHgB –(13. 2-15. 2 g/dL) 13. 5g/dL

SCr-(0. 5-1. 4mg/dL) 0. 7mg/dLHct(40-52%) 39%

Na+(134- 146 mEq/dL) 136 mEq/dLPlts –(140-450 mm3) 300, 000/mm2

Cl-(98-107mEq/dL) 103 mEq/dLWBC –(4. 1-10. 9mm3) 8700/mm2

Bicarb– (22-26 mEq/dL) 24 mEq/dLESR– (<30mm/hr.) 17mm/hr.

Glucose – (65-110mg/dL) 101mg/dLB12 –(223-1132 pg. /ml) 452pg/ml

Ca-(8. 9-10. 4 mEq/dL) 8. 5 mEq/dLfolate– (3. 6-20ng/dL) 6. 4ng/dL

Mag –(1. 6-2. 4mEq/dL)1. 9 mEq/dLFTI –(4-11) 6. 3

Phos –(2. 5-4. 5 mg/dL) 3. 3 mg/dLT3 –(75-220ng/dL) 101ng/dL

Cholesterol-(<200mg/dL) 160mg/dLT4– (4-11mEq/dL) 6. 1 mEq/dL

TSH– (0. 35-6. 2 microU/uL) 2. 0 micro Unit/uLRPR– non-reactive

Radiology Testing

CT scan impression –mild cortical atrophy

Diagnosis

Dementia (senile dementia) is a syndrome rather than a distinct disease entity. It is usually progressive and irreversible. It is characterized by a general decline in cognitive abilities that may include losses of memory, abstract reasoning, judgment, and impulse control, as well as changes in personality. It is usually subtle in onset and often progresses slowly until symptoms are very obvious and profoundly devastating. The three most common dementias are Alzheimer’s disease, multi-infarct dementia, and a mixed Alzheimer’s disease and multi-infarct dementia (Cayton, Graham, & Warner, 2004).

Alzheimer’s disease is sometimes called primary degenerative dementia or senile dementia of the Alzheimer’s type. It accounts for at least 80 percent of all the dementias suffered by the elderly (Whalley, Lawrence, & Breitner, 2009). It is a progressive, irreversible, degenerative neurologic disease of unknown origin that begins insidiously. The highest incidence is among persons 65 years and older with increasing incidence after age 70. The life expectancy following the diagnosis varies from six to twenty years (Whalley, Lawrence, & Breitner, 2009).

The etiology of the disease is unknown, but there are specific neuropathologic and biochemical changes. These include neurofibrillary tangles and senile or neuritic plaques. This neuronal damage occurs primarily in the cerebral cortex and results in decreased brain size. These changes are found to a lesser extent in normal brain tissue of older adults. Cells principally affected by this disease are the ones that use the neurotransmitter acetylcholine. Biochemically, the enzyme active in producing acetylcholine is decreased. Acetylcholine is specifically involved in memory processing (Whalley, Lawrence, & Breitner, 2009).

Clinical manifestation

Symptoms of Alzheimer’s disease are highly variable. Early in the disease, forgetfulness and subtle memory loss occur, but the victim has adequate cognitive function to hide the loss. Social skills and behavior patterns remain intact; problems are difficult to detect on casual observation (Cayton, Graham, & Warner, 2004). With further progression of the disease there is an inability to conceal the deficits. Forgetfulness is manifested in many daily actions. The victim may lose his way in a familiar environment. He may repeat the same stories because he forgets that he told them. Reasoning and reality orientation by caregivers increase the patient’s anxiety without increasing function, because this is also forgotten (Cayton, Graham, & Warner, 2004).

Conversations become difficult because the victim forgets what he was about to say or may not be able to remember words. Ability to formulate concepts and think abstractly disappears (Cayton, Graham, & Warner, 2004). The person can interpret a proverb only in concrete terms. The victim is often unable to appreciate the consequences of his actions and will therefore exhibit impulsive behavior. He will have difficulty with everyday activities such as working simple appliances and handling money (Cayton, Graham, & Warner, 2004).

Personality changes are usually negative. The patient may become depressed, suspicious, paranoid, hostile, and even combative (Whalley, Lawrence, & Breitner, 2009). Progression of the disease intensifies the symptoms. Speaking skills deteriorate to nonsense syllables; agitation and physical activity increase. A voracious appetite often develops because of the high activity level. The patient may wander at night for hours. Eventually he will need help in all areas of personal care including toileting and eating; dysphagia occurs and incontinence develops. The terminal stage may last for months (Cayton, Graham, & Warner, 2004).

Treatment

Treatment for dementia of the Alzheimer’s type will be Aricept 5 mg once daily at bedtime. After four weeks symptoms will be reviewed, and titration to ten mg once daily may be initiated, depending upon results. Maximum dosage of 23 mg daily if needed after three months of 10-mg treatments (Katzung, Mastes, & Trevor, 2012). Follow up in office in four weeks.

Discontinued use of Prevacid, as the medication is implicated in low platelet, WBC, HgB and Hct, calcium, and B12 levels (Brunton, Chabner, & Knollman, 2011).

Follow-up blood work in four weeks, should include CBC with differential, CMP, liver panel, weight, and blood pressure. Patient is borderline hypertensive and increase in fluid volume may correct this issue. Patient should be encouraged to change diet to a high-protein diet with adequate hydration. If platelet count and WBC count continue to be below normal a hematology consult will be discussed. Referral to Alzheimer’s support group will be given to the daughter.

## References

Brunton, L., Chabner, B., & Knollman, B. (2011). Goodman & Gilman’s: The pharmacological basis of therapeutics (12 ed.). McGraw-Hill.

Cayton, H., Graham, N., & Warner, J. (2004). Dementia: Alzheimer’s and other dementias: At your fingertips’ guide (2 ed.). London: Class.

Katzung, B., Mastes, S., & Trevor, A. (2012). Basic & clinical pharmacology (12 ed.). McGraw-Hill.

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