

Disease	Features	Course
<p>Alzheimer's disease (Senile dementia is an older term. May also be called Senile dementia, Alzheimer's type [SDAT])</p>	<p>Most common form of dementia believed to be caused by a lack of chemical in brain causing neurofibrillary tangles, neuritic plaques. One theory is that memory occurs when beta amyloid, a brain protein, triggers a leak of choline from cell membranes. This results in a reduction of acetylcholine production in cells. Choline is a key ingredient of acetylcholine, and helps store and retrieve memories.</p>	<p>Onset age: 60–80</p> <p>Slowly progressive and irreversible. The person has progressive memory loss, behavioral changes, poor judgment, loss of ability for abstract thinking. Progresses to complete disorientation, apathy, loss of speech, and loss of self care ability. Most people die within four to six years after diagnosis, but the illness can last from 3 to 20 years.</p>
<p>Vascular dementia (Multi-infarct dementia)</p>	<p>Vascular lesions cause focal damage. Reduced blood circulation to brain cells caused by arteriosclerosis or atherosclerosis. Second most common form of dementia after Alzheimer's disease. Believed to be caused by a series of strokes that cause a progressive mental decline.</p>	<p>Onset age: 45–70</p> <p>Sudden onset, usually in persons over age 45; more common in men than women; resident may have history of cardiovascular disease. Deterioration is stepwise; uneven decline in skills and gradual personality changes.</p> <p>Resident usually shows focal neurological signs. Outcome depends on rate of brain damage. Persons with a history of CVA have a 9 times greater risk of dementia compared with those who have not had a stroke. Approximately 1 in 4 stroke survivors develop signs of dementia within 1 year.</p>
<p>Huntington's disease</p>	<p>Genetic condition for which testing is available. Inherited from a parent with a gene for the disease. There is a 50% chance of inheriting the gene. Causes a progressive mental decline, speech impairment, muscle rigidity, jerking,</p>	<p>Onset age: 25–45</p> <p>Rate of progression and the age of onset vary from person to person. Average duration 15 years.</p>

	<p>choreiform movements, depression, and ritualistic behavior. Most residents also have mood swings, irritability, difficulty or inability to learn new things, memory loss, difficult decision-making. As the disease progresses, concentration on intellectual tasks becomes difficult. or impossible. Drugs that increase dopamine in brain are being used to reduce involuntary movements with good results. (These are used for symptom control only; they are not a cure.)</p>	
Lewy body dementia	<p>Named for round nerve cell deposits found on autopsy that differ from Alzheimer's deposits. Agitation, delusions, and problems speaking are early symptoms. Causes progressive mental decline, fluctuating alertness and attention span, drowsiness, staring into space for long periods, and visual hallucinations. Motor symptoms are similar to those of Parkinson's disease.</p>	<p>Usually develops later in life than other dementias; typically between ages 68 and 80. Progresses rapidly. The person is generally severely confused within six months of diagnosis.</p>
Parkinson's disease	<p>Dopamine deficiency in brain that causes a progressive mental decline in some persons. Others do not develop mental deterioration. Signs of dementia include memory loss, poor judgment, distractibility, slowed thinking, disorientation, confusion, moodiness, apathy, and lack of motivation.</p>	<p>Dementia develops in approximately 20% of people with Parkinson's, usually those who are diagnosed after age 70. A 10 to 15 year delay from diagnosis and onset of dementia is common. Parkinson's is also caused by Lewy bodies, but occur in a different area of the brain than those seen in Lewy body dementia.</p>
Tertiary syphilis	<p>Untreated syphilis causes neurological problems. The spirochete (bacteria) causes brain damage. The internal organs are also affected. Characterized by formation of gummas, or soft areas of inflammation similar to granulomas. These result from the inability of the</p>	<p>Occurs 1-20 years after initial infection, but can develop up to 50 years later. The person may be highly symptomatic, but the condition is not contagious. No special precautions beyond standard precautions are necessary. The disease is progressively fatal and cannot be cured.</p>

	<p>immune system to clear the organism from the body. Other neurologic signs and symptoms are neuropathic joint disease, loss of sensation, loss of proprioception (fine position sense), personality changes, changes in affect, hyperactive reflexes, and Tabes dorsalis (locomotor ataxia), which results in a shuffling gait. The Argyll-Robertson pupil response is an uncommon diagnostic sign seen in this condition in which the pupils are small and irregular in size and shape. They constrict in response to focusing the eyes, but not to light.</p>	
<p>Creutzfeldt-Jakob disease and other prion diseases</p>	<p>Prions are believed to be the cause of mad cow disease, scrapie, which is seen in sheep and goats, and variant CJD, a mysterious wasting disease seen in elk, deer, and in humans who have ingested infected meat. Fatal familial insomnia is another disease in humans which was recently discovered to be caused by prions.</p> <p>The existence of prions was discovered in the 1980s. Prion is an abbreviation for proteinaceous infectious particle. Prions may be ingested through infected food, such as meat. They can be transmitted only through unclean</p>	<p>Usual onset age: 50–60</p> <p>Rapidly progressive, incurable, and fatal within a short period of time. Symptoms include rapidly progressing dementia, hallucinations, severe memory loss, seizures, loss of motor function, and death within a few months, sometimes less than a few weeks. About 90 percent die within 1 year.</p>
<p>Creutzfeldt-Jakob disease and other prion diseases (cont.)</p>	<p>instruments and body fluids that contain the protein from within the blood or more commonly fluids from the infected brain. Researchers theorize that the prion protein becomes problematic when it ends up in the wrong part of a cell. When this occurs, it binds to mahogunin, a protein that is needed for brain cell survival. This process deprives cells of functional</p>	

	<p>mahogunin, causing them to die. This is believed to be the cause of the neurodegeneration seen in prion diseases.</p>	
<p>AIDS dementia complex (ADC)</p>	<p>Occurs in advancing HIV disease when CD4 counts decrease and immune system destruction is widespread. The changes are called ADC when they are believed to be related to the HIV infection instead of other factors, such as medication side effects or brain infection. Rule out depression, drug side effects, and infectious conditions such as histoplasmosis or toxoplasmosis.</p>	<p>Signs and symptoms occasionally precede a diagnosis of AIDS because persons of nursing home age are not usually tested for HIV infection. Early signs and symptoms often difficult to distinguish from depression.</p> <p>Signs and symptoms include slowed thinking, short attention span, poor concentration, forgetfulness, short- or long-term memory loss, social withdrawal, irritability, apathy, weakness, poor coordination, impaired judgment, problems with vision and personality change. ADC is characterized by profound changes in the person's ability to:</p> <ul style="list-style-type: none"> <li>• Understand</li> <li>• Process and remember information</li> <li>• Behave in a socially acceptable manner</li> <li>• Control emotions</li> <li>• Coordinate muscles and movement</li> </ul>
<p>Lyme disease</p>	<p>The most common vector-borne infection in the United States, Lyme disease is caused by a spirochete. It is increasing in incidence and geographic spread.</p> <p>Lyme is a great masquerader that has been misdiagnosed as many conditions, including schizophrenia, bipolar disorder, anorexia nervosa, OCD, multiple sclerosis and dementia.</p>	<p>Up to 40% of persons with Lyme disease develop neurologic involvement of either the peripheral or central nervous system. Many of these individuals do not get the characteristic bull's-eye rash. Persons with a history of tick bite and those in Lyme-endemic areas should be tested if new, rapid onset dementia develops. Early recognition and treatment are important to preventing this acute, treatable illness from</p>

		becoming a chronic or relapsing condition.
Pick's disease	About 5% of all dementia. Atrophy of frontal and temporal lobes can be seen on brain scan. Similar to Alzheimer's, but not as widespread within the brain.	Onset age 40–60, progressive; similar to Alzheimer's disease. Affects more women than men. Early signs and symptoms occur in emotional and social functioning. The person may experience mood swings, euphoria, disinhibition, and a deterioration in social skills. The resident may be extroverted or withdrawn. He or she may be rude, impatient, aggressive, and make inappropriate comments to others. Sexual behavior may change. The result is often an increased interest in sex or loss of inhibition. The person may overeat, develop pica, or place inappropriate items in the mouth. As the disease progresses, the resident will have difficulty finding words and naming items. Memory loss and apraxia worsen. The resident will have difficulty maintaining a line of thought, be easily distracted and have difficulty maintaining conversation for any length of time.