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DEMENTIA

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ABSTRACT

Dementia, a syndrome with many causes, affects >5 million people in the United States and results in a total annual health care cost between $157 and $215 billion. Dementia is de ned as an acquired deterioration in cognitive abilities that impairs the successful performance of activities of daily living. Episodic memory, the ability to recall events specific in time and place, is the cognitive unction most commonly lost; 10% o persons age >70 years and 20–40% o individuals age >85 years have clinically identifable memory loss. In addition to memory, dementia may erode other mental aculties, including language, visuospatial, praxis, calculation, judgment, and problem-solving abilities. Neuropsychiatric and social deficits also arise in many dementia syndromes, manifesting as depression, apathy, anxiety, hallucinations, delusions, agitation, insomnia, sleep disturbances, compulsions, or disinhibition. T e clinical course may be slowly progressive, as in Alzheimer's disease (AD); static, as in anoxic encephalopathy; or may uctuate rom day to day or minute to minute, as in dementia with Lewy bodies. Most patients with AD, the most prevalent orm o dementia, begin with episodic memory impairment, although in other dementias, such as frontotemporal dementia, memory loss is not typically a presenting feature.

Introduction Dementia syndromes result rom the disruption o specific large-scale neuronal networks; the location and severity o synaptic and neuronal loss combine to produce the clinical features. Behavior, mood, and attention are modulated by ascending noradrenergic, serotonergic,

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and dopaminergic path- ways, whereas cholinergic signaling is critical or atten- tion and memory unctions. T e dementias di er in the relative neurotransmitter deficit profiles; accordingly, accurate diagnosis guides e ective pharmacologic therapy.

AD begins in the entorhinal region o the medial temporal lobe, spreads to the hippocampus, and then moves to lateral and posterior temporal and parietal neocortex, eventually causing a more widespread degeneration. Vascular dementia is associated with ocal damage in a variable patchwork o cortical and sub- cortical regions or white matter tracts that disconnect nodes within distributed networks. In keeping with its anatomy, AD typically presents with episodic memory loss accompanied later by aphasia or navigational prob- lems. In contrast, dementias that begin in rontal or subcortical regions, such as frontotemporal dementia (FTD) or Huntington's disease (HD), are less likely to begin with memory problems and more likely to present with di culties with judgment, mood, executive control, movement, and behavior.

Lesions of frontal-striatal pathways produce specific and predictable effects on behavior. T e dorsolateral pre rontal cortex has connections with a central band o the caudate nucleus. Lesions o either the caudate or dorsolateral pre rontal cortex, or their connecting white matter pathways, may result in executive dysfunction, manifesting as poor organization and planning, decreased cognitive exibility, and impaired working memory. T e lateral orbital rontal cortex connects with the ventromedial caudate, and lesions of this system cause impulsiveness, distractibility, and disinhibition. T e anterior cingulate cortex and adjacent medial prefrontal cortex project to the nucleus accumbens, and interruption of this system produces apathy, poverty o speech, emotional blunting, or even akinetic mutism. All corticostriatal systems also include topographically organized projections through the globus pallidus and thala- mus, and damage to these nodes can likewise reproduce the clinical syndrome of cortical or striatal injury.

Materials and Methods: The causes of dementia. T e single strongest risk actor or dementia is increas- ing age. T e prevalence o disabling memory loss increases with each decade over age 50 and is usu- ally associated with the microscopic changes o AD at autopsy. Yet some centenarians have intact memory function and no evidence of clinicallysigni cantdementia. Whether dementia is an inevitable consequence of normal human aging remains controversial. T e requency of each condition depends on the age group under study, access o the group to medical care, country o origin, and perhaps racial or ethnic back- ground. AD is the most common cause o dementia in Western countries, accounting or more than hal of all patients. Vascular disease is considered the second most requent cause or dementia and is particularly common in elderly patients or populations with limited access to medical care, where vascular risk actors are undertreated. Of en, vascular brain injury is mixed with neurodegenerative disorders, making it difficult, even or the neuropathologist, to estimate the contribution o cerebrovascular disease to the cognitive disorder in an individual patient. Dementias associated with Parkinson's disease (PD) are common and may develop years affer onset o a parkinsonian disorder, as seen with PD-related dementia (PDD), or can occur concurrently with or preceding the motor syndrome, as in dementia with Lewy bodies (DLB). In patients under theageo 65,F Drivals AD as the most common cause of dementia. Chronic intoxications, including those resulting rom alcohol and prescription drugs, are an important and of en treatable cause of dementia. Other disorders listed in able 21-1 are uncommon but important because many are reversible. T e classiffication of dementing illnesses into reversible and irreversible disorders is a

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useful approach to di erential diagnosis. When e ective treatments or the neurodegenerative conditions emerge, this dichotomy will become obsolete.

In a study o 1000 persons attending a memory disorders clinic, 19% had a potentially reversible cause of the cognitive impairment and 23% had a potentially reversible concomitant condition that may have contributed to the patient's impairment. T e three most common potentially reversible diagnoses were depression, normal pressure hydrocephalus (NPH), and alcohol dependence; medication side effects are also common and should be considered in every patient. Subtle cumulative decline in episodic memory is a common part of aging. This frustrating experience, of en the source o jokes and humor, is refferred to as benign forgetfulness of the elderly. Benign means that it is not so progressive or serious that it impairs reasonably success ul and productive daily unctioning, although the distinction between benign and more significant memory loss can be difficult to make. At age 85, the average person is able to learn and recall approximately one-hal of the items (e.g., words on a list) that he or she could at age 18. A measurable cognitive problem that does not seriously disrupt daily activities is of en refferred to as mild cognitive impairment (MCI). Factors that predict progression rom MCI to an AD dementia include a prominent memory deficit, family history of dementia, presence of an apolipoprotein ε4 (Apo ε4) allele, small hippocampal volumes, an AD-like signature of cortical atrophy, low cerebrospinal fluid Aβ, and elevated tau or evidence of brain amyloid deposition on positronemissiontomography (PE) imaging.

T e major degenerative dementias include AD, DLB, F D and related disorders, HD, and prion diseases, including Creutz eldt-Jakob disease (CJD). T ese disorders are all associated with the abnormal aggregation of a specific protein: $A\beta_{42}$ and tau in AD; α-synuclein in DLB; tau, AR DNA-binding protein of 43 kDa (DP-43), or fused in sarcoma (FUS) in FTD; hunting- tin in HD; and mis olded prion protein (PrP^{SC}) in CJD.

CLINICAL DIFFERENTIATION OF THE MAJOR DEMENTIAS

DISEASE	FIRST SYMPTOM	MENTAL STATUS	NEUROPSYCHIATRY	NEUROLOGY	IMAGING
AD	Memory loss	Episodic memory loss	Irritability, anxiety, depression	Initially normal	Entorhinal cortex and hippocampal atrophy
FTD	Apathy; poor judgment/insight, speech/language; hyperorality	Frontal/executive and/or language; spares drawing	Apathy, disinhibition, overeating, compulsivity	May have vertical gaze palsy, axial rigidity, dystonia, alien hand, or MND	Frontal, insular, and/or temporal atrophy; usually spares posterior parietal lobe
DLB	Visual hallucinations, REM sleep behavior disorder, delirium, Capgras syndrome, parkinsonism	Drawing and frontal/executive; spares memory; delirium-prone	Visual hallucinations, depression, sleep disorder, delusions	Parkinsonism	Posterior parietal atrophy; hippocampi larger than in AD
CJD	Dementia, mood, anxiety, movement disorders	Variable, frontal/executive, focal cortical, memory	Depression, anxiety, psychosis in some	Myoclonus, rigidity, parkinsonism	Cortical ribboning and basal ganglia or thalamus hyperintensity on diffusion/FLAIR MRI
Vascular	Often but not always sudden; variable; apathy, falls, focal weakness	Frontal/executive, cognitive slowing; can spare memory	Apathy, delusions, anxiety	Usually motor slowing, spasticity; can be normal	Cortical and/or subcortical infarctions, confuent white matter disease

Abbreviations: AD, Alzheimer's disease; CBD, cortical basal degeneration; CJD, Creutzfeldt-Jakob disease; DLB, dementia with Lewy bodies; FLAIR, fuid-attenuated inversion recovery; FTD, frontotemporal dementia; MND, motor neuron disease; MRI, magnetic resonance imaging; PSP, progressive supranuclear palsy; REM, rapid eye movement.

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Treatment. T e major goals of dementia management are to treat reversible causes and to provide comfort and support to the patient and caregivers. reatment o underlying causes includes thyroid replacement or hypothyroidism; vitamin therapy or thiamine or B_{12} deficiency or for elevated serum homocysteine; antimicrobials or opportunistic in ections or antiretrovirals or HIV; ventricular shunting or NPH; or appropriate surgical, radiation, and/or chemotherapeutic treatment or CNS neoplasms. Removal of cognition impairing drugs or medications is requently useful. If the patient's cognitive complaints stem from a psychiatric disorder, vigorous treatment of this condition should seek to eliminate the cognitive complaint or confirm that it persists despite adequate resolution of the mood or anxiety symptoms. Patients with degenerative diseases may also be depressed or anxious, and those aspects o their condition of en respond to therapy. Antidepressants, such as selective serotonin reuptake inhibitors (SSRIs) or serotonin-norepinephrine reuptake inhibitors (SNRIs) which eature anxiolytic properties but few cognitive side effects, provide the mainstay of treatment when necessary. Anticonvulsants are used to control seizures. Levetiracetam may be particularly use ul, but there have as yet been no randomized trials or treatment o AD-associated seizures.

Agitation, hallucinations, delusions, and con usion are di cult to treat. T ese behavioral problems represent major causes or nursing home placement and institutionalization. Be ore treating these behaviors with medications, the clini- cian should aggressively seek out modi able environmental or metabolic actors. Hunger, lack o exercise, toothache, constipation, urinary tract or respiratory in ection, electro- lyte imbalance, and drug toxicity all represent easily cor-rectable causes that can be remedied without psychoactive drugs. Drugs such as phenothiazines and benzodiazepines may ameliorate the behavior problems but have untoward side e ects such as sedation, rigidity, dyskinesia, and occa- sionally paradoxical disinhibition (benzodiazepines). Despite their un avorable side e ect pro le, second-generation anti- psychotics such as quetiapine (starting dose, 12.5–25 mg daily) can be used or patients with agitation, aggression, and psychosis, although the risk pro le or these compounds is signi cant. When patients do not respond to treatment, it is usually a mistake to advance to higher doses or to use anticholinergic drugs or sedatives (such as barbiturates or benzodiazepines). It is important to recognize and treat depression; treatment can begin with a low dose o an SSRI (e.g., escitalopram, starting dose 5 mg daily, target dose 5–10 mg daily) while monitoring or e cacy and toxicity. Some- times apathy, visual hallucinations, depression, and other psychiatric symptoms respond to the cholinesterase inhibitors, especially in DLB, obviating the need or other more toxic therapies.

Cholinesterase inhibitors are being used to treat AD (done- pezil, rivastigmine, galantamine) and PDD (rivastigmine). Recent work has ocused on developing antibodies against $A\beta_{42}$ as a treatment or AD. Although the initial randomized controlled trials ailed, there was some evidence or e cacy in the mildest patient groups. There fore, researchers have begun to ocus on patients with very mild disease and asymptomatic individuals at risk or AD, such as those who carry autoso- mal dominantly inherited genetic mutations or healthy elders with CSF or amyloid imaging biomarker evidence supporting presymptomatic AD. Memantine proves use ul when treating some patients with moderate to severe AD; its major bene t relates to decreasing caregiver burden, most likely by decreas- ing resistance to dressing and grooming support. In moderate to severe AD, the combination o memantine and a cholines- terase inhibitor delayed nursing home placement in several studies, although other studies have not supported the e cacy o addingmemantinetotheregimen.

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A proactive strategy has been shown to reduce the occur- rence o delirium in hospitalized patients. T is strategy includes requent orientation, cognitive activities, sleep enhancement measures, vision and hearing aids, and correction of dehydration.

Nondrug behavior therapy has an important place in dementia management. T e primary goals are to make the patient's life comfortable, uncomplicated, and safe. Preparing lists, schedules, calendars, and labels can be help ul in the early stages. It is also use ul to stress amiliar routines, walks, and simple physical exercises. For many demented patients, memory or events is worse than their ability to carry out routine activities, and they may still be able to take part in activities such as walking, bowling, dancing, singing, bingo, and gol . Demented patients often object to losing control over amiliar tasks such as driving, cooking, and handling finances. Attempts to help or take over may be greeted with complaints, depression, or anger. Hostile responses on the part o the caregiver are counterproductive and sometimes even harmful. Reassurance, distraction, and calm positive statements are more productive in this setting. Eventually, tasks such as nances and driving must be assumed by others, and the patient will con orm and adjust. Sa ety is an important issue that includes not only driving but controlling the kitchen, bathroom, and sleeping area environments, as well as stairways. These areas need to be monitored, supervised, and made as safe as possible. A move to a retirement complex, assisted-living center, or nursing home can initially increase confusion and agitation. Repeated reassurance, reorientation, and care ul introduction to the new personnel will help to smooth the process. Providing activities that are known to be enjoyable to the patient can be of considerable benefit.

T e clinician must pay special attention to rustration and depression among amily members and caregivers. Caregiver guilt and burnout are common. Family members of en eel overwhelmed and helpless and may vent their rustrations on the patient, each other, and health care providers. Caregivers should be encouraged to take advantage of day-care facilities and respite services. Education and counseling about dementia are important. Local and national support groups, such as the Alzheimer's Association (www.alz.org), can provide considerable help.

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ОҒРИҚ СИНДРОМИ СИНДРОМИНИНГ ПАТОГЕНЕТИК МЕХАНИЗМИ ВА КЛИНИК ВАРИАНТЛАРИ. Журнал вестник врача, 1(1 (98)), 144-146

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