**Depression vs Dementia**

Depression in any age group is often associated with cognitive disturbance. In the elderly, the cognitive disturbance is often severe enough to appear like a dementia, causing many to call this a pseudodementia. Depressive symptoms and cognitive disturbance can also be caused by medications such as tranquilizers or pain medications, medical illness such as hypothyroidism, stroke (over 50% of patients with anterior, dominant hemisphere CVA’s develop depression), or Parkinson’s disease. This is why medication history and medical review is needed.

It is widely held that core depressive symptoms like sleep disturbance, anorexia, anhedonia, negativism, and tearfulness will persist in all major depressions, and should be looked for as part of the confirmation of depression as opposed to another cause. Generally, sleep disturbance in depression fits the pattern of short sleep latency (falls asleep immediately), but interrupted sleep with early morning rising (wakes for the final time at about 4am), with concomitant daytime sleepiness.

Cognitive disturbance is generally different from Alzheimer’s Dementia. Usually patients with depression make errors due to errors of omission - they answer, “I don’t know”, whereas patients with dementia make errors of commission - they give a wrong answer. Depressed patients become low risk takers (they won’t venture an answer). Depressed patients are often fully oriented but make errors on concentration tasks, like serial subtraction, or errors on effortful tasks. Depressed patients sometimes show the reverse pattern of memory loss than in dementia, with forgetfulness of both recent and remote items, as opposed to just recent memory loss.

Atypical depression is responsive to somatic treatments for depression, both medication and ECT. It is also helped by reducing environmental stresses, helping patients develop better coping styles, or working through conflict areas like pathological mourning.

One caveat is that depression often causes an excess disability. In terms of dementia, the confusion is often worse when depressed. In the case of medical problems, even mild problems like hypertension can lead to disability from weakness, headaches, and vague symptomatology that defy treatment. Often with depression in dementia, cognitive function improves with treatment of depression, but does not go away fully.

Depression, at least the first episode, is usually triggered by a psychosocial event, like a loss or onset of chronic medical illness. One presumes that there is a biological predisposition for depression, but the biological cascade of events that leads to the catecholamine disregulation that causes depression, is usually triggered by an external psychiatric pathogen (stress). It is usually necessary to help reduce or eliminate the stress or impact of stress through psychotherapy. Psychotherapy can take many forms and have different goals, depending upon the psychological theory used to conceptualize the current problem.

Dementia in late life is common. Dementia is defined as the absence or reduction of intellectual faculties in consequence of known, organic brain disease. It is said that the prevalence of AD, the most common cause of late-life dementia, doubles every 5 years after the age of 65. From an epidemiologic study from Harvard about 10 years ago, 3% of the age range 65-74 had probable AD, 15% of the age group 75-84, and 47% of those over age 85.
Usually patients and families are less concerned about the memory loss itself, which many people wrongly assume is inevitable (Old Timers Disease), than the non-cognitive aspects of dementia (behavior problems, management issues).

There are many possible causes of dementia with their own risk factors, course, risk of co-morbid psychiatric problems and, in some cases, treatments to improve function and/or reduce decline. There may be more than one potential cause for dementia that co-exists. The only current FDA approved class of medications for AD is the cholinesterase inhibitors that are known to improve cognition and slow decline. The currently used cholinesterase inhibitors are donepazil (Aricept), galantamine (Excelon), and rivastigmine (Reminyl). Tacrine (Cognex) is no longer in use. AD typically progresses at a rate of about 2 MMSE points per year decline, and in most cases it takes more than a decade to go from diagnosis to 24-hour custodial care or death.

The type of memory problem associated with AD mirrors the brain functions associated with the areas where plaques and tangles are greatest (frontotemporal and occipital). Usually there are significant memory problems and disorientation (hippocampus), progressive aphasia (parietotemporal), visual-spatial (occipital) problems, and frontal lobe features.

As with most brain diseases, dementia is associated with an increased association with depression, psychosis, anxiety, impulse disorders, and non-specific agitation (wandering, inappropriate vocalizations, pacing, appropriating, etc.). Appropriate psychiatric medications decrease the primary psychiatric syndromes (depression, anxiety, psychosis, and aggression), but do little for the non-specific behavioral symptoms that still must be approached through behavioral management. Often strategies as simple as reducing noise or having a fixed schedule or having time outs are enough to reduce agitation. It is not uncommon to see denial or projection as a defense against loss of self-esteem. Rather than acknowledge deficits in oneself, patients may blame others for hiding things, or frankly deny problems that appear obvious.

When beliefs become entrenched (delusional), antipsychotics are needed, but caution should be taken in using older neuroleptics, like phenothiazines or haloperidol due to the risk of tardive dyskinesia (20% incidence rate per year in the elderly). A little known fact about AD is that learning still occurs. It has been found that stimulus-response learning (classical conditioning) remains relatively intact, and that implicit memory (non-verbalized or out-of-awareness knowledge) remains relatively intact. Explicit memory (test taking cortical skills) is of course, extremely impaired.