Integrated perspectives of Neuro-cognitive Disorders and Trauma

Presented by:

NICOLE ABSAR, M.D
Diplomate, Behavioral Neurology & Neuro-Psychiatry.
Diplomate, American Board of Psychiatry & Neurology.
Cingulate Cortex, Amygdala, Hippocampus and Stress
Early Presentations of Cognitive Disorders

- Memory: Forgetfulness, misplacing objects
- Executive: Poor Attention, Difficulty with money management, problem solving, decision making
- Visuo-spatial: Getting lost during driving or lost in familiar places, problem with ADL's (dressing, bathing, cooking)
- Language: Word finding difficulties, semantic meaning
- Visual Recognition: problem with face recognition, object, recognition
- Personality change & behavioral change
- Delusions: Capgrass, spousal infidelity, persecutory
- Mood, Motivation, Motor/memory: Falls, gait, tremor,
**Stress reaction to amygdala:** Amygdala is responsible for survival-related threat identification, plus tagging memories with emotion. After trauma the amygdala can get caught up in a highly alert and activated loop during which it looks for and perceives threat everywhere.

**Effect of Cortisol in hippocampus:** An increase in the stress hormone glucocorticoid kills cells in the hippocampus, which renders it less effective in making synaptic connections necessary for memory consolidation. This interruption keeps both the body and mind stimulated in reactive mode as neither element receives the message that the threat has transformed into the past tense.

**Results of Chronic Stress:** The constant elevation of stress hormones interferes with the body’s ability to regulate itself. The sympathetic nervous system remains highly activated leading to fatigue of the body and many of its systems, most notably the adrenal.
Genetic Predisposition

Amygdala-HPA-Cortical Pathway

Environmental Risks

Cingulate Cortex, Amygdala, Hippocampus and Stress
• Report by Joel Pieper, MD, MS, of University of California, San Diego.

• In this study, 89 current or former members of the military with mild traumatic brain injury. Using standard symptom scale ratings, 29 people were identified with significant PTSD. The rest had mild traumatic brain injury without PTSD.

• The subjects with mild traumatic brain injury and PTSD had 6 percent overall larger amygdala volumes, particularly on the right side, compared to those with mild traumatic brain injury only.
- Memory is one of the cognitive domain
- Other domains also need to be affected
- Loss of previous level of functioning, unable to self care
- Not due to medical, psychiatric, or drug problems

Memory Loss Alone is not Dementia
• Amnesic Syndrome (MEMORY DISORDERS)
• Aphasic Syndrome (language Disorder)
• Apraxic Syndrome (earned motor acts /perceptory)
• Agnostic syndrome (facial /visual recognition problems)
• Dys-executive Syndrome (problem solving, abstract thinking, judgment, mental flexibility, multi-tasking, impulsivity)
• Compartmental Syndrome (apathy, amotivational, mutism)
Acute: Clinical Course Abrupt from Hours to Days:  
---Delirium or Acute Confusional State (Abrupt Change in Mental Status)

Sub Acute: Clinical Course Weeks to Months:  
---Reversible Dementia( due to treatable causes of dementia)

Chronic Clinical Course & Progressive:  
---Dementia Syndrome
What is Delirium?

- Delirium is a Global Cognitive Disorder of Abrupt In Onset
- Delirium is an Acute Medical Emergency State
- It is often reversible, correctable, preventable
- Delirium is an abrupt change in the level of consciousness/sensorium, Abrupt change in the mental status and behavior
• Infections:
  Systemic: pneumonia, UTI, Septicemia
  Neurological: meningitis/encephalitis

• Metabolic:
  (dehydration/electrolytes D/O, liver/kidney D/O)

• Toxic:
  Street Drugs: Substance intoxication/withdrawal
  Prescription Drugs: anti-cholinergic/cold meds/
  or low albumin or drug-drug interactions
Clinical Presentation of Dementias

- First Presentation: Memory
- First presentation: Personality & behavioral change/depression
- First Presentation: Gait, Falls/Spatial
- First presentation: language
- First Presentation: REM sleep change, Visual Hallucinations/
- First Presentation: Movement D/O
Rapidly Progressive Dementia

- TRAUMATIC BRAIN INJURY
- CNS INFECTION
- INFLAMMATION/AUTO-IMMUNE
- METABOLIC/ENDOCRINE
- VASCULAR
- Paraneoplastic Limbic Encephalitis
- TOXIC
- OBSTRUCTIVE
- Psychiatric (Depression/Mood Disorder)
Depression Syndrome of Dementia

Primary Depression:
Melancholic with Psycho-motor Symptoms

- Secondary Depression:
- Micro-Vascular Depression:
  Micro-Angiopathic White Matter Disease due to anoxia, HTN, DM
- Macro-Vascular Depression:
  Left Hemispheric, Left Basal Ganglia, Cerebellar Cognitive affective syndrome (CCAS)
- Inflammatory:
  SLE, Sarcoid, Auto-Immune
- HPA Axis:
  Hypo-thyroidism, Hypo-parathyroidism
  Toxic (Heavy metals, Drugs)
  Infectious, Neoplastic, Degenerative, Traumatic
• Always rule out Involuntary Emotional Expression Disorder (IEED) from Mania

• Rule Out Secondary Mania

• Check for Infectious Mania: Neurosyphilis, HIV, Lyme, Metastatic abscess

• Check for Auto-Immune Mania (SLE, Sarcoid)

• Check for Vascular Mania:
  R Hemispheric Stroke,
  Basal Ganglia Disease (PD, HD, WD, Fabrys,)
  R/O Neoplastic/Traumatic/Degenerative causes

Mania Syndrome of Dementia
<table>
<thead>
<tr>
<th></th>
<th>Normal Aging</th>
<th>Preclinical AD</th>
<th>MCI</th>
<th>Alzheimer’s Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathological Diagnosis</strong></td>
<td>Normal</td>
<td>AD</td>
<td>AD</td>
<td>AD</td>
</tr>
<tr>
<td><strong>ADL</strong></td>
<td>Intact</td>
<td>Intact</td>
<td>Intact</td>
<td>Impaired</td>
</tr>
<tr>
<td><strong>Clinical symptoms &amp; signs of cognitive impairment</strong></td>
<td>None</td>
<td>None</td>
<td>Subjective memory loss</td>
<td>Cognitive impairment</td>
</tr>
<tr>
<td><strong>Tangles in entorhinal cortex/hippocampus</strong></td>
<td>Few to many (increase with age)</td>
<td>Many</td>
<td>Many</td>
<td>Many</td>
</tr>
<tr>
<td><strong>Plaques in neocortex</strong></td>
<td>None or few</td>
<td>Moderate to frequent</td>
<td>Moderate to frequent</td>
<td>Moderate to frequent</td>
</tr>
</tbody>
</table>

Normal Aging: None or few
Preclinical AD: Moderate to frequent
MCI: Moderate to frequent
Alzheimer’s Dementia: Moderate to frequent
Presentations of Alzheimer’s Disease

- Temporo-limbic (Hippocampal) w/ early memory symptoms
- Parieto-Occipital w/ early visuo-perceptory/apraxia (Posterior Cortical Atrophy)
- Frontal Variant w/ early behavioral/language symptoms
• Early impairment in regulation of personal & social conduct
  • Early emotional blunting
  • Early loss of insight
• Instrumental functions of perception, spatial skills, praxis, and memory are intact or relatively well preserved.

• Clinical presentations: one involving behavioral and personality change, and the other involving language impairment.

• Supportive features
  Behavioral disorder:
  • Mental rigidity and inflexibility
  • Distinctibility and impersistance
  • Hyperorality and dietary changes
  • Perseverative and stereotyped behavior
  • Utilization behavior

  Language presentations of frontotemporal dementia usually appear later in the disease and after the behavior changes, but they eventually converge.

  Speech and language:
  • Altered speech output
  • Aspontaneity and economy of speech
  • Press of speech
  • Stereotype of speech
  • Echolalia
  • Perseveration
  • Mutism

  Physical signs:
  • Primitive reflexes
  • Incontinence
  • Akinesia, rigidity, and tremor
  • Low and labile blood pressure

Fronto-temporal Dementia (FTD)
• Personality Change
• Apathy/Disinhibition
• Decline in personal hygiene
• Mental rigidity and inflexibility
• Distractibility & impersistence
• Hyperorality
• Perseverative and stereotyped behavior
• Utilization behavior

FTD: Early Behavioral variant.
Clinical Diagnosis of LBD

- Core Features:
  - Fluctuation of cognition/sensorium with pronounced variation in attention and alertness (fluctuating confusion)
  
  Recurrent Visual hallucinations that are well formed and detailed

  Spontaneous motor features of Parkinsonism
Clinical Diagnosis of LBD

Supportive features:

- Falls
- Sympathetic dysautonomia (Autonomic dysfunction w/syncope, BP fluctuations)
- Neuroleptic sensitivity
- Systematized delusions
- Hallucinations in other modalities
- REM sleep behavioral disorder
- Depression
PET Imaging: AD vs FTD
Subcortical Dementias (Syndrome of 3 M’s)

• Mood & Motivation: (Personality/apathy/depression)
• Motor/Movement: tremor/gait
• Memory: Retrieval type Short term memory and executive dysfunction.
Subcortical Dementia

- **Vascular Dementia.**

  WHITE MATTER DEMENTIA (DEMYELINATING DEMENTIA):
  - Dementia due to white matter disease including M.S.

- **Basal Ganglia Dementia**
  - Parkinson's Disease
  - Huntington's Disease
  - Wilson's Disease
  - Progressive Supra nuclear Palsy (PSP)

- **CEREBELLAR DEMENTIA:**
  - CEREBELLAR COGNITIVE AFFECTIVE SYNDROME (C.C.A.S)
    - EXECUTIVE DYSFUNCTION
    - SPATIAL DYSFUNCTION
    - LINGUISTIC
    - PSYCHIATRIC: MOOD /PERSONALITY DISTURBANCES

Nicole M. Absar M.D
- NEUROLOGICAL INTERVENTION
- PSYCHIATRIC INTERVENTION.
- COGNITIVE HEALTH:
  - NUTRITION: MIND DIET
  - PHYSICAL EXERCISE, WALKING, AEROBIC CARDIAC EXERCISE
  - Yoga & Mindfulness.
  - COGNITIVE STIMULATION

INTEGRATED NEUROCOGNITIVE CARE
• A diet that is good for the heart is also good for the brain.

• Mediterranean diet has become well known for its cardiac benefits. With a focus on fresh fruits and vegetables, beans, fish, and olive oil, the Mediterranean diet has been shown to reduce the risk of heart disease as well as overall mortality.

• A eating plan called the DASH diet (Dietary Approaches to Stop Hypertension) was developed by the National Institutes of Health, has been found effective in reducing hypertension.

• In addition to the cardiac benefits, both the DASH diet and the Mediterranean diet have been shown to improve cognition; however, neither were developed to slow neurodegeneration.
Team at Rush University Medical Center, headed by Martha Clare Morris, Sc.D worked to create a hybrid Mediterranean-DASH Intervention for Neurodegenerative Delay diet (MIND diet).

This hybrid diet has the basic components of the Mediterranean and Dash diets, emphasizing natural plant-based foods and limited intake of animal-based and high saturated fat foods.

It also includes recommendations for specific foods, like leafy greens, berries, and nuts that have been scientifically shown to slow cognitive decline. These foods contain high levels of antioxidants that help slow down the breakdown of neuronal cell loss.

The diet also recommends avoiding or eating only rarely foods like red meat, butter, fried foods and sweets. Recent research has shown that the MIND diet is more effective at reducing cognitive decline than either the Mediterranean or DASH diets alone.
But Diet alone can’t prevent Alzheimer’s disease. Genetics and other modifiable risk factors including smoking, exercise, level of education and mental health also play a role.

However MIND diet helped slow the rate of cognitive decline and protect against various neurocognitive disorders including Alzheimer’s disease, regardless of other risk factors.

The diet should be complimented with structured daily physical exercise targeting cardiovascular health, as well as mental and cognitive exercise.
Yoga and mindfulness have proven benefits to improving neurochemistry, especially in the pre-frontal cortex of the brain.

Pre-frontal cortex controls our day to day physical and mental activities such as planning, organizing, problem solving, awareness of our needs, attention, multi-tasking, improvement mood and anxiety.
The word ‘yoga’ is derived from the Sanskrit word for ‘union’—meaning a united body, mind and soul.

In modern medicine and neurology, we now know that the ultimate therapeutic approach is one built on an integrated mind, body and brain connection.

Yoga bridge the mind and body and brain with breathe.
Risk factors for Impaired cognitive health

- Obesity and Sedentary lifestyle and Lack of Exercise
- Smoking, drug abuse
- Cardiovascular disease
- Diabetes
- Hypertension
- Hyperlipidemia
- Stroke
- Head Injury
- Stress & Mood Disorder
- Sensory deprivation
• Periodic wellness visit for health screening
• Management of chronic medical issues like diabetes, hypertension, hyperlipidemia, Cardiac disease.
• Avoid prescription medications with toxic side effects on memory, sleep, and executive functions like benzodiazepines, Anti-histamines and Anti-cholinergic and sedatives and opiates.
• Reduce risk for brain injuries due to falls and other head injuries.
• Limit Alcohol and avoid drinking while on CNS depressant drugs.
• Quit Smoking and drug abuse
• Sleep Hygiene : sleeping 7-8 hours each night.
Yoga and Dementia

- Mood and Motivation
- Attention
- Sleep
- Anxiety
- Obsessive compulsion
- Pain
- Pleasure
HPA axis activated by Stress
- Stress activate sympathetic nervous System
- Pranayama breathing activates the parasympathetic nervous system.

The parasympathetic response calms the body and neutralizes the stress and anxiety triggered by an overactive sympathetic nervous system.
In the end, only 3 things matter:

how much you loved, how gently you lived, and how gracefully you let go of things not meant for you.

-Buddha

rawforbeauty.com
Nicole Absar, MD
Board Certified in American Board of Psychiatry & Neurology

617-669-6816  nmabsar@aol.com

Consultant & Educator
Specializing in Neurocognitive Disorders