OVERVIEW OF DEMENTIA AND THE DIAGNOSTIC PROCESS

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AIM OF THIS TALK

- Overview of how people are referred to be assessed for dementia
- Overview of how people present with the various forms of dementia
- Overview of the tests and investigations for the various forms of dementia
- Overview of (some of) the various forms of Dementia
- A word on Capacity
- Some Real Life Cases
- Happy to take questions then
- Very happy to take emails etc later if people have particular questions they want to ask

WHAT IS DEMENTIA?

- Dementia is a syndrome in which there is deterioration in memory, thinking, behaviour and the ability to perform everyday activities.
- Worldwide, around 50 million people have dementia, and there are nearly 10 million new cases every year.
- Alzheimer disease is the most common form of dementia and may contribute to 60–70% of cases.
- Dementia is one of the major causes of disability and dependency among older people worldwide.
- Dementia has a physical, psychological, social, and economic impact, not only on people with dementia, but also on their carers, families and society at large.

WHAT DEMENTIA ISN'T

- Here are a few things that dementia **isn't**:
  - it isn't a single disease
  - it isn't a normal part of ageing
  - it isn't the same experience for everyone
  - It isn't something that only older people get, it can happen in younger people too
- 9 out of 10 people **don't** get dementia

THE HISTORY OF DEMENTIA

- Alzheimers Disease: Named for Alois Alzheimer
- German psychiatrist and pathologist
- First published case of presenile dementia
- Patient called Auguste Deter (1850-1906) in Frankfurt asylum
  - She had a lack of short term memory
  - At post mortem discovered amyloid plaques and neurofibrillary tangles – hallmarks of Alzheimer's disease
  - Died at the age of 55 – would be diagnosed with early onset if she were alive today

HOW DO PATIENTS WITH DEMENTIA PRESENT?

- Commonest presentation is to the GP with memory trouble
- Sometimes patients with more rapid/severe symptoms may present to other services like psychiatry
- GP might refer on to a specialist service such as Geriatrics/Neurology/Psychiatry depending on what the patient presents with and what age they are
- Occasionally patients only present when a crisis has happened to the emergency department
  - In that case they often can end up under the care of a general medical team, or the speciality that happened to be taking patients for that day
- Sometimes the diagnosis is extremely obvious and apparent at an early stage
- Sometimes the diagnosis is not obvious, may require multiple referrals and investigations before it is reached
- Occasionally a definitive diagnosis is not reached, but a only a high suspicion of what a diagnosis is

HOW DO DOCTORS EXAMINE PEOPLE?

- GPs typically have between 7 and 15 minutes to see patients
- If someone comes into an emergency department
  - ED SHO/Reg/Consultant might have 5-30mins with a patient
  - If referred medically: Medical SHO/Reg has 45minutes/1 hour with a patient
- If they end up admitted medically consultant will spend ~15mins with patient on post take ward round (recommended British guidelines at least half an hour), then daily following this
  - Then it depends on investigations and if they come to a diagnosis during inpatient stay or are referred to a different service for inpatient/outpatient review
- In an Outpatient clinic it depends on the type of clinic as to how long a doctor has with the patient. Anywhere from 15 minutes to 60 minutes
- Specialist memory services try and spend much longer with patients and their families

STANDARD DOCTORS INTERVIEW AND EXAM

- All doctors are taught a standard interview and exam technique in medical school, using the following format:
  - Presenting Complaint
  - History of Presenting Complaint
  - Past Medical History
  - Past Surgical History
  - Medications
  - Family History
  - Social History
  - Review of Systems
  - Collateral History - NB

EXAMINATION

- Vital Signs:
  - Heart Rate, Blood Pressure, Oxygen Saturations, Weight, Height, BMI, Temperature
- Full Physical Examination includes:
  - Cardiovascular, Respiratory, Gastrointestinal and Neurological Examination
- Based on the information you may have gathered you may do other exams:
  - Breast, Thyroid, Rheumatological etc

FOCUSED MEMORY HISTORY AND EXAM

- In the history taken from the patient and a collateral , trying to establish:
  - Are they having issues with their short or long term memory?
  - How long have the issues been going on for?
  - Is their speech affected?
  - How is their eyesight and hearing?
  - Has their level of function been affected?
  - Are they having any issues with executive function?
  - Have they had any issues with their driving?
  - Would you trust them in an airport on their own?
  - Are they having any hallucinations?
  - Are they having any other medical issues?
  - Can they be confused about where they are?

FOCUSED MEMORY HISTORY AND EXAM *CONT….*

- Are they losing interest in self care?
- Are they asking repetitive questions, or engaging in repetitive behaviour?
- Are they forgetting to buy food or groceries?
- Are they becoming more socially withdrawn?
- Are they getting lost, or wandering?
- Are they becoming confused about the time of the day?
- Are they becoming more irritable or agitated?
- Are they missing events or appointments?
- Are they currently going through the menopause?
- Are they depressed?

FOCUSED MEMORY HISTORY AND EXAM *CONT…*

- Past Medical History
  - Have they any neurological conditions?
  - Have they had a previous stroke?
  - Have they any significant traumatic brain injury in the past/history of concussion?
  - Have they any significant history of infection?
  - Have they any history of cancer?
  - Have they any history of significant hospital admission, any admission to ICU?
  - Have they ever had a general anaesthetic?
  - Have they a significant psychiatric history?

FOCUSED MEMORY HISTORY AND EXAM *CONT…*

- Medications:
- Are they on medications which can impact on memory?
  - Anticholinergics
  - Chemotherapy agents
  - Psychiatric Medicines
  - Thyroid Replacement Therapy
  - Treatment for HIV

FOCUSED MEMORY HISTORY AND EXAM *CONT…*

- Family History:
  - Is there a history of Dementia within the family?
  - Is there any family history of psychiatric illness?
  - Is there a family history of suicide in later life, or people with behavioural changes in later life?
  - Any family history of any illness at all?

SOCIAL HISTORY

- Can they read and write?
- What's the highest level of education they've obtained?
- Is English their first language?
- What is their occupation?
- Do they smoke? How much and for how long?
- Do they drink alcohol? Have they a history of alcoholism?
- Have they a history of drug abuse?
- Have they a history of risky sexual behaviour?

SYSTEMIC REVIEW

- In the systemic review we ask leading questions for a brief overview of overall health, looking for any other health problems
- Often answers here can point towards diagnoses, or even offer clues as to an alternate diagnosis

EXAMINATION

- Looking for signs of various dementias
  - General appearance, wearing clothes with stains on them, poorly applied makeup, signs of not having washed or bathed, lack of interest in personal appearance
  - Do they struggle with interpreting commands on the exam?
  - Are they obviously poorly sighted or struggling with their hearing?
  - Do they exhibit signs of parkinsons disease?
    - Small shuffling steps, tremor, muscle rigidity, slow movements, quieter, face less expressive, more prone to falls
  - Do they show language difficulties? And if so what type?
  - Can they look up?

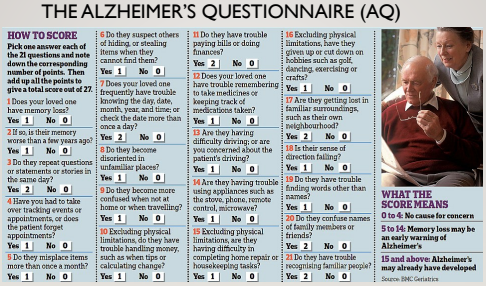
INVESTIGATIONS

- Blood tests
  - Looking for treatable causes of memory trouble
  - Deficiencies in b12, folate, Iron can cause some memory issues
  - Thyroid function tests: Underactive thyroids can cause issues with memory
  - FBC, U and E, LFTs, Bone Profile: Checking for any other abnormality in routine bloods
  - STI Screen can be performed where you might suspect syphilis or AIDS dementia
- ECG:
  - Should be done as standard, important for when/if considering treatment.

INVESTIGATIONS

- Neuropsychological Assessment:
- Pen and Paper type memory testing that are designed to show you if someones memory is working and see what domains people are struggling with eg language, recall, executive function, visuospatial ability
- Standard easy to administer ones include the MMSE and MOCA
- A very quick one that can be given to a collateral to fill out is the AQ (Alzheimers Questionnaire)
- Trained neuropsychologists/consultants/ANPs/OTs might administer much more in-depth tests such as the R-Bans, CAMCOG, ACE-R, WIMS-III, DWR etc
- The score you get on these tests does not give you a diagnosis of dementia, but it informs the clinical decision, based on how it correlates with the history, exam and findings from other investigations

THE ALZHEIMER'S QUESTIONNAIRE (AQ)



HOW TO SCORE

Pick one answer each of the 21 questions and note down the corresponding number of points. Then add up all the points to give a total score out of 27.

1 Does your loved one have memory loss?
Yes 1 No 0

2 If so, is their memory worse than a few years ago?
Yes 1 No 0

3 Do they repeat questions or statements or stories in the same day?
Yes 2 No 0

4 Have you had to take over tracking events or appointments, or does the patient forget appointments?
Yes 1 No 0

5 Do they misplace items more than once a month?
Yes 1 No 0

6 Do they suspect others of hiding, or stealing items when they cannot find them?
Yes 1 No 0

7 Does your loved one frequently have trouble knowing the day, date, month, year, and time; or check the date more than once a day?
Yes 2 No 0

8 Do they become disoriented in unfamiliar places?
Yes 1 No 0

9 Do they become more confused when not at home or when travelling?
Yes 1 No 0

10 Excluding physical limitations, do they have trouble handling money, such as when tips or calculating change?
Yes 1 No 0

11 Do they have trouble paying bills or doing finances?
Yes 2 No 0

12 Does your loved one have trouble remembering to take medicines or keeping track of medications taken?
Yes 1 No 0

13 Are they having difficulty driving; or are you concerned about the patient's driving?
Yes 1 No 0

14 Are they having trouble using appliances such as the stove, phone, remote control, microwave?
Yes 1 No 0

15 Excluding physical limitations, are they having difficulty in completing home repair or housekeeping tasks?
Yes 1 No 0

16 Excluding physical limitations, have they given up or cut down on hobbies such as golf, dancing, exercising or crafts?
Yes 1 No 0

17 Are they getting lost in familiar surroundings, such as their own neighbourhood?
Yes 2 No 0

18 Is their sense of direction failing?
Yes 1 No 0

19 Do they have trouble finding words other than names?
Yes 1 No 0

20 Do they confuse names of family members or friends?
Yes 2 No 0

21 Do they have trouble recognising familiar people?
Yes 2 No 0

WHAT THE SCORE MEANS

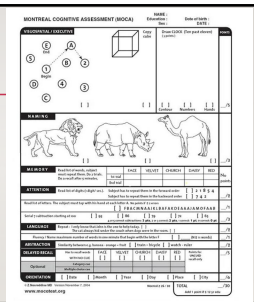
0 to 4: No cause for concern

5 to 14: Memory loss may be an early warning of Alzheimer's

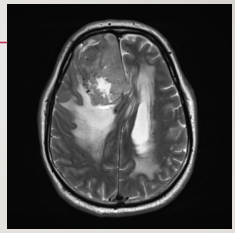
15 and above: Alzheimer's may already have developed

Source: BMC Geriatrics

NEUROPSYCHOLOGICAL ASSESSMENT



INVESTIGATIONS: RADIOLOGY



- Gold Standard is an MRI with coronal cuts
- You want to get SOME kind of imaging of the brain, in many instances CT brain is acceptable
- Main reason you want brain imaging is to out-rule a brain tumour
- Once this has been outruled you can then look for other findings which correlate with a dementia diagnosis
- CT gives you less information than an MRI, but is still helpful

INVESTIGATIONS: RADIOLOGY

- What are you looking for on a scan?
  - Generalised and localised: i.e. atrophy of particular lobes, in particular paying attention to the temporal lobes
  - Dilatation of the ventricles
  - Hippocampal atrophy: Graded using the Medial Temporal lone Atrophy Scale
    - There is also the Global Cortical Atrophy Scale and the Posterior Cortical Atrophy Scale
  - Gradient Echo: Used to assess for Cerebral Amyloid Angiopathy

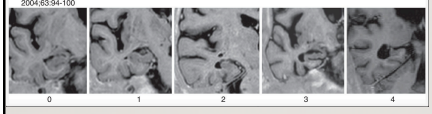
INVESTIGATIONS: RADIOLOGY *CONT …*

- White Matter disease and signs of previous strokes:
  - White matter disease may be secondary to vascular risk factors such as hypertension, diabetes etc or may have discrete areas representing previous strokes
- Hydrocephalus:
  - Can lead to issues with cognition eg normal pressure hydrocephalus
- Infections
  - Eg PML in HIV/AIDS patients, rare infections like TB
- Disease specific signs: Eg Hummingbird sign, Hot Cross Bun Sign

Medial temporal lobe atrophy

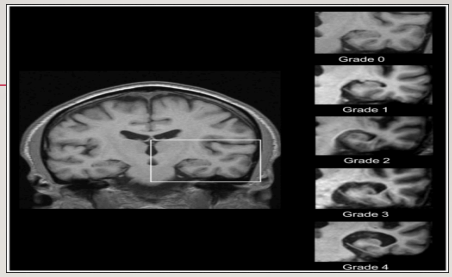
Score	Amplitude of the choroidal fissure	Amplitude of the temporal horn	Height of hippocampus
0	Normal	Normal	Normal
1	Slightly widened	Normal	Normal
2	Moderately widened	Slightly widened	Slightly reduced
3	Significantly widened	Moderately widened	Moderately reduced
4	Significantly widened	Significantly widened	Significantly reduced

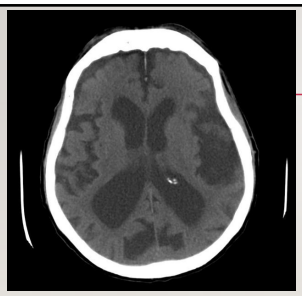
Korf E. et. Al Medial temporal lobe atrophy on MRI predicts dementia with mild cognitive impairment. *Neurology* 2004;63:94-100



MEDIAL TEMPORAL LOBE ATROPHY SCALE

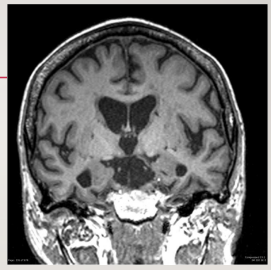
MEDIAL TEMPORAL LOBE ATROPHY

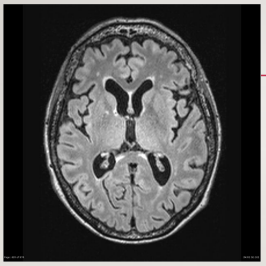




CT SCAN SHOWING ATROPHY AND DILATATION

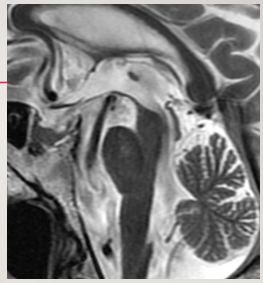
MRI – T1 AXIAL SHOWING HIPPOCAMPAL ATROPHY

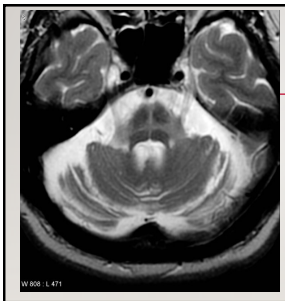




AXIAL FLAIR SHOWING GLOBAL ATROPHY

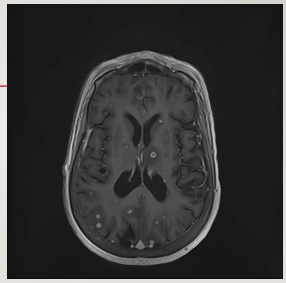
HUMMINGBIRD SIGN OF PSP





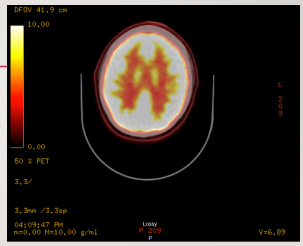
HOT CROSS BUN SIGN OF MSA

MILIARY TB – POST CONTRAST MRI



INVESTIGATIONS: RADIOLOGY

- PET Brain:
- FDG-PET available in some centres in Ireland



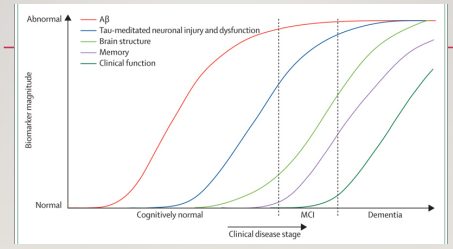
INVESTIGATIONS: RADIOLOGY: PETS *CONT…*

- Essentially the opposite idea to Whole body PETs:
  - You are looking to see if any areas of the brain are hypometabolic, rather than in cancer where the tumours are typically metabolically active
  - PET-CT uses radiolabeled glucose analogue FDG to measure glucose metabolism, which indicates levels of neuro-synaptic activity
  - FDG = Fluorodeoxyglucose
  - Typically a piece of software such as NeuroQ will be able to give you a numerical value of the activity of the brain in lots of different areas when compared to a normal person
  - A score of less than -2.5 in any area is considered abnormal and suggestive of neurodegeneration
  - Alzheimers classically is seen affecting the posterior cingulate gyrus first

INVESTIGATIONS: CSF BIOMARKERS

- By using a lumbar puncture to take a large sample (5ml) of cerebrospinal fluids we can analyse this fluid to help us diagnose Alzheimers Disease
- We look at three proteins:
  - Amyloid Beta 42:40
  - Total Tau
  - Phosphorylated Tau
- If the Tau proteins are high and the amyloid is low it is a very sensitive test for Alzheimers disease
- Don't recommend doing it in older people routinely (>75 typically) as its diagnostic value goes down with advancing age
- Can be done anywhere but needs to be collected in a special sample jar and sent to St James's

PATH THAT A TYPICAL ALZHEIMERS DISEASE TAKES



DEMENTIA

- Name the types of Dementia
- Not a trick question

TYPES OF DEMENTIA

- Alzheimers Disease
- Posterior Cortical Atrophy
- Vascular Dementia
- Fronto-Temporal Dementia
- Primary Progressive Aphasia
  - Can be either an Alzheimers type or FTD type
- Amyloid Angiopathy
- Progressive Supranuclear Palsy
- Lewy-Body Dementia
- AIDS Dementia
- Alcohol Dementia
- Parkinsons Dementia
- Memory issues/Dementias associated with long term psychiatric illnesses eg bipolar
- Probably more that I have forgotten

COGNITIVELY NORMAL

- More and more people worried about their memory presenting to memory clinics/GPs
- Peoples memories DO change when they get older
- Very common for us all to get "delayed information processing speed" ie it takes us longer to take in new information and remember stuff from our memories
- THIS IS NORMAL
- "You can't run as fast as when you were in your 20's, can you?"
- Often co-existing psychiatric/psychological issues, or people with family histories eager to get checked out

DEPRESSION

- People with depression have presented to the memory clinic with memory loss
- Complain of fatigue, difficulties with focus and concentration
- Typically in younger people
- Does happen in older people as well

MILD COGNITIVE IMPAIRMENT

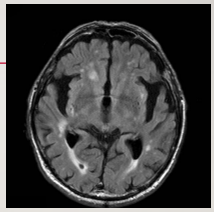
- Not a disease
- It's a way of characterising patients who clearly have a memory problem and have poor test scores, but aren't functionally compromised
- Roughly 50% will go on to develop dementia, 50% won't
- Most important thing is that they do not have functional impairment
- Most of the people who have mild cognitive impairment have an Alzheimers PATHOLOGY, but have not yet gone on to develop Alzheimers DEMENTIA

ALZHEIMER'S DEMENTIA

- Caused by Alzheimers Disease
- Most Common Cause of Dementia
- More prevalent as you get older, can happen in younger people
  - Inherited forms associated with mutations in APP, PSEN1 and PSEN2
  - Mean age in this group 46 years, earliest on average PSEN1 age 43
  - Down Syndrome higher likelihood of developing it early due to additional copy of APP on their additional Chromosome 21
  - Less than one percent of all cases of AD
- Doubles in prevalence every 5 years from 65
- 1 in 3 in their 80s, 1 in 2 in their 90's will get it
- Aetiology of it still poorly understood – we are still not sure why it happens and what causes it

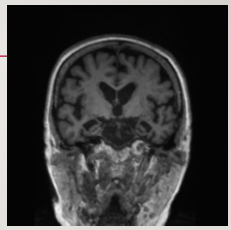
ALZHEIMER'S DEMENTIA

- Cardinal Feature
  - Progressive Short term memory impairment
  - Declarative memory typically affected (memory of events which took place at a time and place)
  - Progresses over time until all facets of memory are affected
- Executive Function/Judgement problem solving can also be affected
- Patients lose insight over time
- May suffer from the behavioural and psychological symptoms of dementia over time
- Very heterogenous illness
  - Onset typically slow and insidious
  - Can happen quickly though
- Can also develop praxic difficulties, sleep disturbance, seizures, motor signs



ALZHEIMER'S DISEASE

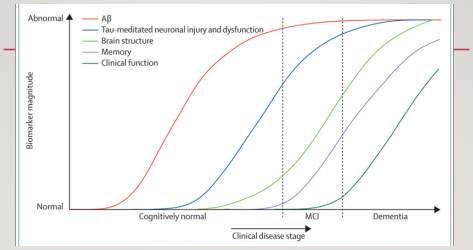
- Pathologically:
  - Neuritic Plaques secondary to Extracellular Amyloid Beta Deposition
  - Neurofibrillary plaques secondary to Intracellular phosphorylated tau deposition
- On Neuroimaging
  - Gold Standard is MRI
  - Coronal Cuts to look for Hippocampal atrophy
  - Also looking for atrophy elsewhere (particularly temporal lobes) and vascular changes as well
  - PET scan, shows hypometabolism. Classically the posterior cingulate gyrus is involved, but can be hypometabolic else/everywhere



ALZHEIMER'S DISEASE

- Can check CSF biomarkers
  - Looking at Amlyoid Beta, Total Tau and Phosphorylated Tau
  - If A-Beta is low, and the two Taus are high its characteristic of an AD type process
- Blood tests
  - Check for reversible causes, b12, folate, thyroid function
  - Also check for suspect medications

PATH THAT A TYPICAL ALZHEIMER'S DISEASE TAKES



TREATMENT OF ALZHEIMER'S

- No cure
- Cholinesterase Inhibitors
  - Donepezil
  - Memantine
  - Rivastigamine Patch

WHAT ELSE TO DO?

- Make a will
- Make an enduring power of attorney
  - Nominate someone to take over your financial decisions should you become incapacitated
- Exercise
- Reduce Red Meat
- Mediterranean Diet (colourful, leafy fruit and veg)
- Reduce Stress
- Stop Smoking
- Reduce alcohol intake

WHAT ELSE TO DO?

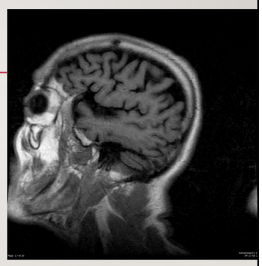
- Driving:
  - You HAVE to inform your insurance company
  - You HAVE to apply for a new license
  - Need an on road driving assessment
  - People with AD can drive! Are often very safe in the initial to middle stages, most will give it up themselves
- Work
  - People who are working should continue doing so if they can
  - Need support and structure
  - Taking things away from people with dementia is not the right thing to do unless absolutely necessary

WHAT ELSE TO DO?

- Relatives/Carers:
  - May be eligible for carers allowance (means tested)
  - Also will be eligible for fuel/heating grant (once off payment every June of ~€1500)
- Advise them to contact Alzheimer's Society
- Look for supports in their area
- Can be a good idea to contact public health nurse

VARIANTS OF ALZHEIMER'S

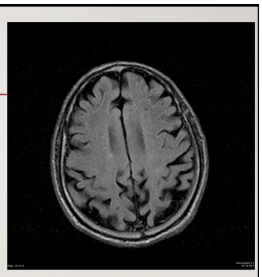
POSTERIOR CORTICAL ATROPHY



- Typically present with visual and praxic difficulties secondary to progressive cortical visual impairment
- Relative sparing of anterograde memory function, speech and nonvisual language functions, executive functions, and behavior and personality
- Eventually will develop into an Alzheimers like process
- Neuroimaging usually shows predominant occipitoparietal or occipitotemporal atrophy, hypometabolism, or hypoperfusion

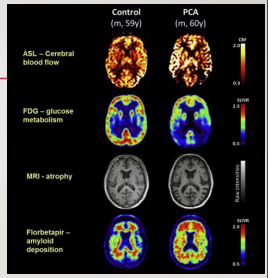
POSTERIOR CORTICAL ATROPHY

- Space perception deficit
- Simultanagnosia (ie, the inability to integrate a visual scene despite adequate visual acuity to resolve individual elements)
- Object perception deficit
- Constructional dyspraxia
- Environmental agnosia
- Oculomotor apraxia (the inability to direct gaze accurately to a new target)
- Dressing apraxia



POSTERIOR CORTICAL ATROPHY

- Left/right disorientation
- Acalculia
- Limb apraxia
- Apperceptive prosopagnosia
- Agraphia
- Homonymous visual field defect
- Finger agnosia
- Optic ataxia (the inability to reach accurately under visual guidance)
- Alexia



PRIMARY PROGRESSIVE APHASIA

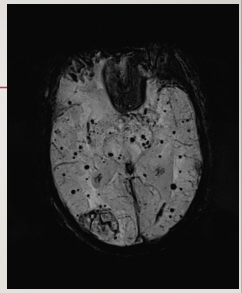
- Progressive degeneration in language difficulty with relative sparing of memory and other functions
- Can be either due to Alzheimers or Fronto-Temporal Pathology
- The Alzheimers form is more common as AD is more common than FTD
- Eventually progress on to a full dementia picture
- Three classifications nonfluent, semantic, or logopenic
- Logopenic tends to be AD pathology

LOGOPENIC VARIANT PPA

- The logopenic variant of PPA features impaired single-word retrieval and repetition with errors in speech and naming, but spared single-word comprehension and object knowledge, spared motor speech, and absence of agrammatism
- Slow rate of speech is primarily due to word-finding pauses, rather than difficulties with word production, articulation, or apraxia of speech
- The speech of logopenic variant PPA has been described as "empty," in the sense that a patient might tell a story vaguely or with words that profoundly lack descriptive detail.
- Phonological short-term memory deficits in logopenic variant PPA manifest as difficulty with long, grammatically complex sentences, although single-word repetition is spared.

CEREBRAL AMYLOID ANGIOPATHY (CAA)

- CAA is a disorder caused by accumulation of amyloid in cerebral vessels, and it leads to multiple lobar hemorrhages, microbleeds, or infarctions. CAA is prevalent in those with AD, and dementia in these individuals has been traditionally ascribed to AD.
- However, extensive CAA can also cause ischemic white matter damage, a complication that is presumably related to diffuse narrowing of penetrating cortical vessels by amyloid deposits



LEWY BODY DEMENTIA

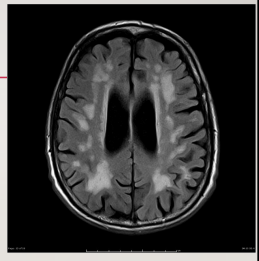
- Lewy body dementia, also known as dementia with Lewy bodies, is the second most common type of progressive dementia after Alzheimer's disease dementia. Protein deposits, called Lewy bodies, develop in nerve cells in the brain regions involved in thinking, memory and movement (motor control).
- In addition to dementia, distinctive clinical features include: visual hallucinations, parkinsonism, cognitive fluctuations, dysautonomia, REM sleep behaviour disorders, syncope and neuroleptic sensitivity.
- Treatment is with rivastigamine/donepezil etc
- Avoid neuroleptics!
- No specific neuroimaging with Lewy Body Disease

VASCULAR DEMENTIA

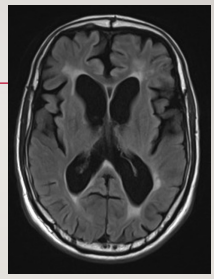
- Vascular dementia is a general term describing problems with reasoning, planning, judgment, memory and other thought processes caused by brain damage from impaired blood flow to your brain.
- You can develop vascular dementia after a stroke blocks an artery in your brain, but strokes don't always cause vascular dementia. Whether a stroke affects your thinking and reasoning depends on your stroke's severity and location. Vascular dementia can also result from other conditions that damage blood vessels and reduce circulation, depriving your brain of vital oxygen and nutrients.
- Factors that increase your risk of heart disease and stroke — including diabetes, high blood pressure, high cholesterol and smoking — also raise your vascular dementia risk.

CLINICAL FEATURES

- What the previous slide meant basically was that over time you get damage to the different areas of the brain from either sudden or progressive ischaemia causing memory issues due to the damage caused
- Very Diverse and heterogeneous effects



DIFFERENCE BETWEEN AD AND VASC

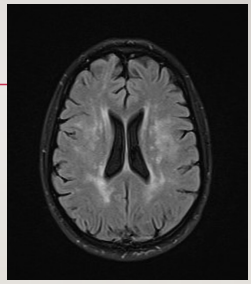


- Often Co-Exist
- AD very classically is rapid short term forgetfulness
- Vasc can be like that too, but tend to be patchy memory problems across multiple domains
- AD is thought of as being gradual, Vascular in stepwise progression
- Not always the case for both of them!
- This is a 55 year old man with vascular dementia where he has significant atrophy and ventricular dilation secondary to his vascular disease

VASCULAR DEMENTIA

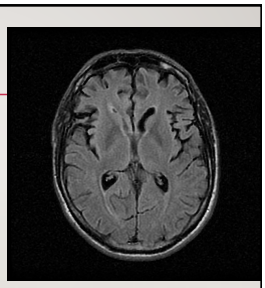
- Few different diagnostic criteria
- NINDS-AIREN criteria
- AHA Criteria
- You need neuroimaging and neuropsych testing
- CT/MRI looking for ischaemic changes
- Neuropsych: You want to check memory function across multiple domains, including immediate and delayed memory, attention, language, praxis and executive function
  - Typically display a patchy, and dysexecutive memory. Might have a terrible short term memory, and be horribly disorganized, but some stuff DOES stick

CADASIL



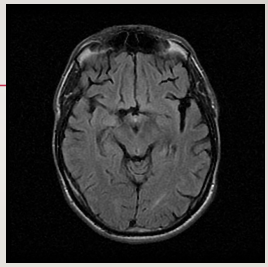
- Cerebral autosomal dominant arteriopathy (CADASIL) is a disorder caused by mutations in the NOTCH3 gene on chromosome 19 that produces subcortical infarcts and leukoencephalopathy.
- Patients develop a subcortical Vascular Dementia syndrome in the fifth to seventh decades, suffering recurrent strokes and ischaemia.

FRONTO-TEMPORAL DEMENTIA



- Frontotemporal dementias (FTD) are a group of clinically and neuropathologically heterogeneous neurodegenerative disorders characterized by prominent changes in social behavior and personality or aphasia accompanied by degeneration of the frontal and/or temporal lobes.

FTD



- 2 Broad Groups:
- Behavioural Variant FTD
- Language Prominent FTDs (sometimes classed as part of primary progressive aphasia)

BEHAVIOURAL VARIANT FTD

- The classical FTD – Picks Disease
- Disinhibition – Examples of disinhibition or socially inappropriate behavior include touching or kissing strangers, public urination, and flatulence without concern. Patients may make offensive remarks or invade others' personal space. Patients with FTD may exhibit utilization behaviors, such as playing with objects in their surroundings or taking others' personal items.
- Apathy and loss of empathy – Apathy manifests as losing interest and/or motivation for activities and social relationships. Patients may participate less in conversations and grow passive. Apathy is mistaken frequently for depression, and patients are often referred for psychiatric treatment early in the disease course.

BEHAVIOURAL VARIANT FTD

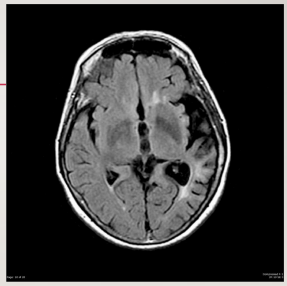
- As patients lose empathy, caregivers may describe patients as cold or unfeeling towards others' emotions. Degeneration of right orbital frontal and anterior temporal regions may drive the loss of sympathy and empathy
- Hyperorality – Hyperorality and dietary changes manifest as altered food preferences, such as carbohydrate cravings, particularly for sweet foods, and binge eating. Increased consumption of alcohol or tobacco may occur. Patients may eat beyond satiety or put excessive amounts of food in their mouths that cannot be chewed properly. They may attempt to consume inedible objects. This behavior correlates with right orbitofrontal, insular, striatal and hypothalamic degeneration
- Compulsive behaviors – Perseverative, stereotyped, or compulsive ritualistic behaviors include stereotyped speech, simple repetitive movements, and complex ritualistic behaviors such as hoarding, checking, or cleaning. Other behaviors traditionally associated with obsessive compulsive disorder, such as hand-washing and germ phobias, are generally absent. Patients with FTD can develop a rigid personality, rigid food preferences, and inflexibility to changes in routine.

BVFTD

- 15-20% develop Motor Neuron Disease (MND)
- Also associated with Corticobasal Syndrome and Progressive Supranuclear Palsy
- CSF biomarkers in FTD are normal, or else you might see elevation in tau and p-tau
- May see frontal/temporal atrophy on neuroimaging

PRIMARY PROGRESSIVE APHASIA- FTLD

- Non-Fluent Variant FTLD
- Semantic Variant FTLD



PPA-FTLD

- **Nonfluent variant PPA** — The core feature of nonfluent variant PPA is a motor speech deficit characterized by effortful production of the linguistic units of sound (phonemes). Although word finding is a common complaint across all PPA subtypes, the characteristic that distinguishes the nonfluent variant from other forms of PPA is articulatory difficulty. This is composed of effortful, halting speech with inconsistent speech-sound errors and distortions and agrammatism in language production
- At the bedside, apraxia of speech can be tested by having the patient repeat a word such as "caterpillar" or "artillery" rapidly and repeatedly, since these words are particularly challenging to articulate. Vowel distortions and difficulty pronouncing such words iteratively are characteristic of speech apraxia. These deficits correspond anatomically to the peak site of atrophy within the left inferior frontal gyrus and posterior fronto-insular atrophy.
- While comprehension is typically spared for single words and simple sentences, patients often have difficulty with complex sentences, particularly those with complex syntax, such as using the passive voice or multiple dependent clauses
- Social comportment, memory, visual spatial skills, and other cognitive abilities are typically preserved at the time of presentation While patients usually retain some insight, they may seem inappropriately unconcerned
- Some patients go on to develop behavioral alterations or symptoms of motor neuron disease (MND) or corticobasal degeneration (CBD)

PPA-FTLD

- **Semantic variant PPA** — The core features of semantic variant PPA are impaired single-word comprehension and object naming in the setting of preserved fluency, repetition, and grammar.
- Word-finding difficulty, especially for low-frequency items, is the earliest symptom, and there is progressive loss of knowledge of the characteristics of objects. Early on, patients may exhibit a marked discrepancy between the ability to understand complete sentences, which tends to be preserved, and the ability to understand single object words, which is impaired
- As the disease progresses, comprehension becomes more globally impaired
- Patients may also exhibit surface dyslexia or dysgraphia, in which words with irregular spellings (eg, yacht, colonel, tissue) are mispronounced or misspelled because the correct pronunciation or spelling relies on semantic knowledge.

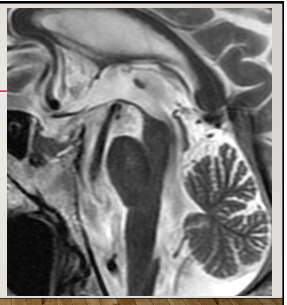
PPA-FTLD – *CONT…*

- At the bedside, the clinician can ask the patient to draw animals such as a turtle, dog, and bird; those with semantic variant PPA frequently make drawings of animals lacking defining features (eg, no shell for a turtle), demonstrating loss of semantic detail around a given animal. In patients with right greater than left temporal pole involvement, facial recognition of famous people may be impaired in the early stages of the disease
- Although asymmetric atrophy of the temporal poles is the rule in the early stages of semantic variant PPA, eventually the disease spreads to the contralateral side. With bilateral involvement, there is emergence of behavioral symptoms such as behavioral rigidity, parsimony of speech, loss of empathy for others, and hypergraphia.
- As disease spreads from anterior temporal lobes to mesial temporal cortices, episodic memory may decline, while visuospatial skills and executive function remain relatively intact

PROGRESSIVE SUPRANUCLEAR PALSY

- Symptoms and signs of Parkinsonism (ie, tremor, bradykinesia, rigidity, and postural instability) can be prominent in neurodegenerative disorders other than idiopathic Parkinson disease, particularly in atypical parkinsonian disorders, which include corticobasal degeneration, multiple system atrophy, and progressive supranuclear palsy.
- Progressive supranuclear palsy (PSP), also known as Steele Richardson Olszewski syndrome, is an uncommon but not rare parkinsonian syndrome.
- Characteristic features of PSP and its variants include vertical supranuclear gaze palsy, postural instability with unexplained falls, akinesia, and cognitive dysfunction.

HUMMINGBIRD SIGN OF PSP



CBD – CORTICOBASILAR DEGENERATION

- The classic description of CBD is that of a progressive asymmetric movement disorder characterized by various combinations of akinesia, rigidity, dystonia, focal myoclonus, ideomotor apraxia, and alien-limb phenomena.
- However, a wider range of clinical presentations is increasingly apparent, including onset with cognitive or behavioral abnormalities.

CBD

- Motor Features:
  - Limb rigidity (57 and 85 percent)
  - Bradykinesia or clumsy limb (48 and 76 percent)
  - Postural instability (41 and 78 percent)
  - Falls (36 and 75 percent)
  - Abnormal gait (33 and 73 percent)
  - Hyperreflexia (30 and 50 percent)
  - Axial rigidity (27 and 69 percent)
  - Tremor (20 and 39 percent)
  - Limb dystonia (20 and 38 percent)
  - Myoclonus (15 and 27 percent)

CBD

- Other problems:
  - Behavioural Disturbance
  - Cortical Dysfunction
  - Cognitive Impairment
  - Oculomotor Dysfunction
  - Speech Disturbance
  - Apraxia
  - Aphasia
  - Alien Limb Phenomenon

CBD

- No formal diagnostic criteria
- Based on clinical and neurological exam
- Neuroimaging may be normal or may see atrophy
- No/poor response to levo/carbidopa (should see a response to extrapyramidal features)

CAPACITY

- Do people with Dementia have capacity?

CAPACITY

- It depends!
- The result of your MMSE might not reflect your capacity!
- Situation Specific
  - Testamentary Capacity
  - Financial Capacity
- Four tenets of Capacity
  - Can they understand the information presented about the decision?
  - Can they retain this information?
  - Can they weigh up this information?
  - Can they communicate this decision (even if it is through someone else)

CAPACITY

- Should be assessed by more than one person ideally
- Should be done on a number of occasions
- Model of how we assess and administer will change when new legislation is enacted with patients having advocates etc but although this is law it hasn't been implemented yet, framework not established

CASES

CASE 1

- 53 year old woman
- Left school at 9, but attended night classes
- Non-smoker, seldom drinker
- Driving, no issues
- 1 year history of short-term memory complaints
- Attended psych due to depression, depression resolved but memory issues persist
- MMSE 20/30 psych assessment
- MOCA 12/30 psych assessment

CASE 1

Subjective Hx

- I have a problem
- Can't remember where we live now, keep thinking we live in old house
- Can't remember day/date
- "I've gone bad with names"
- Forget things quickly
- "Someone will ask me to do things and I'll forget them while doing them"
- Family Hx:
  - Mother RIP 47 "Alcoholism and Cancer"
  - Father RIP 57 "Depression, lost will to live"

CASE 1

Exam

- Struggled to remember what age she was
  - Tried to count the years from 1962
- Little bit fatuous, nice lady though, engaged appropriately
- Normal neuro exam, struggled with commands
- Normal cardio/resp exam
- Mildly elevated BP 175/88

CASE 1

Collateral Hx

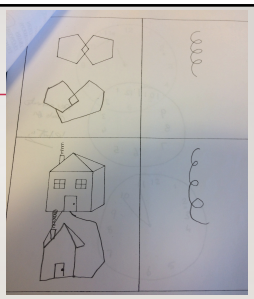
- 2 year Hx
- Lot of presssure on them as they lost their house
  - Pressure now gone, sold house and are renting
- Short term memory not the same
  - Has improved recently
  - Very repetitive
  - Struggling with day/date/time
- Mood normalised
- Function normal
  - He always cooked, cleaned, did housework
  - Works on a community employment scheme

CASE 1

Neuropsych

- MMSE 17/30 (0/3 Recall)
- CAMCOG 63/107 (Memory 9/27)
- DWR Verbal Recall 0/10, Verbal Recognition 4/10
  - (should be over 3 on recall, 10 on recognition)

CASE 1

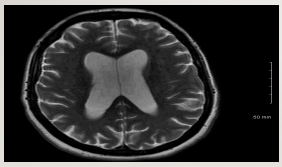


CASE

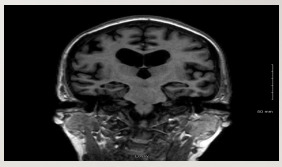
- Consultant Led Consensus Meeting
  - Very Concerning for Alzheimers Disease
- MRI – Global Atrophy
- Very young woman, want to be as sure
  - FDG-PET
  - CSF Biomarkers

CASE 1

AXIAL



CORONAL



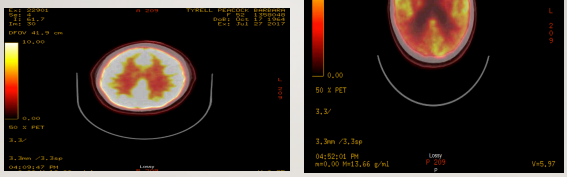
PET SCAN

- "There is unequivocal brain atrophy as evidenced by generalised ventriculomegaly and widening of the cortical sulci"
- Standard region of interest analysis quantified multiple areas of profound deviation from normal standards including the right visual association area. Please see saved images.
- Impression: "The appearances are consistent with an intrinsic neurodegenerative process predominantly involving the right parietal temporal and visual association cortex, posterior cingulate and precuneus. There are some abnormality in the left side as described. The findings were discussed directly with the referring clinician. The appearances favour Alzheimer's dementia"

PET SCAN

CASE 2

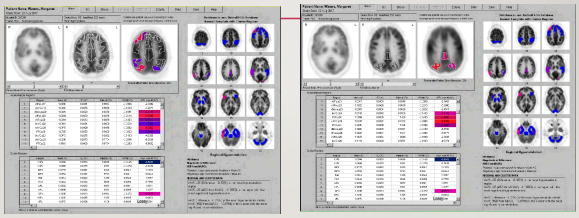
NORMAL PET



NEUROQ

SERIES 1

SERIES 2



CSF BIOMARKERS

General Immunology	
Total Tau	(H) 750.0
Amyloid Beta 1-42	681.9
Phosphorylated Tau	* (H) 103.1
Microbiology	
CSF Microscopy and Culture	

DIAGNOSIS

ALZHEIMERS DISEASE

- Started patient on Donepezil (ECG was normal)
- Gave advice on keeping physically, cognitively and socially active
- Dietary Advice
- Advised re: will and EPOA
- Make contact with PHN
- Talk to Employer
- Can apply for carers allowance
- Disability Grant

DRIVING

- All people diagnosed with AD have to inform insurers and apply for a new license
  - Change of circumstance
- Advise On Road Driving Assessment

CASE 2

- 56 year old woman
- Non-smoker, seldom drinker
- Junior Cert Education
- PMHx Hypothyroidism, post natal depression
- Nil else of note
- No FHx

CASE 2

Subjective History

- 1 ½ year Hx
- Unable to find car in car park
  - When she found her car, couldn't figure out how to use ticket machine
  - Couldn't understand how to put token into machine
- Complaining of issues with vision
- Had eyes checked multiple times, no problems
- Struggling to dress herself
- Worsening short term memory, had a very good memory
- Formerly excellent multi-tasker, now unable
- Not remembering birthdays etc
- Struggling with following recipes
- Very worried

CASE 2

Collateral Hx

- 18/12 Hx
- Worsening Short term memory
- Needs to be reminded of appointments constantly
- Worsening handwriting
- Difficulty using hob, dishwasher etc
- Difficulty using phone
- Thought it was her eyesight and mood at first as increasingly anxious

CASE 2

Examination

- Very well presented, appropriate
- Articulated problems very well
- Mild WFD
- Struggled with Neurological Exam commands
- Past-pointing bilaterally
- Dysdiadokinetic bilaterally
- Very dyspraxic, furniture crawling
- Normal Cardiovascular and Resp Exam

CASE 2

Neuropsych

- Very poor globally.
  - ACE-R was 37/100.
  - CAMCOG 45/107 with a memory composite of 3/22.
  - MMSE (serial 7's and world) was 16/30
  - DWR verbal recall was 0/10, verbal recognition was 7/10.

CASE 2

Consensus Meeting

- MRI Brain Mild Global Atrophy
- Very Concerning Presentation
  - Neurodegenerative Disorder
  - Aetiology as yet unknown
  - Likely Alzheimers Disease or Variant
- PET and CSF Warranted
- Asked Dr Hutchinson (Neurology) for review
- Met with patient and husband, discussed same, happy to proceed
- Patient had put herself off road due to concerns about visuospatial ability

CASE 2

Neurology Assessment

- Detailed assessment by Dr Hutchinson
  - Significant visuospatial problems
  - Simultagnosia, optic apraxia, optic ataxia
  - Alcalculia, dysgraphia, dyslexia
- Lumbar Puncture Preceded PET
  - Totally fine after LP
  - CSF Biomarkers indicative of AD
- PET Scan

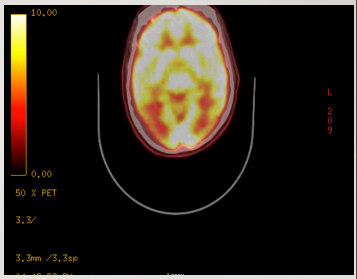
CSF BIOMARKERS

CSF

LABORATORY	17/10/2016 12:38
General Immunology	
Total Tau	(H) 1,820.0
Amyloid Beta 1-42	689.6
Phosphorylated Tau	* (H) 141.3
Microbiology	
CSF Microscopy and Culture	

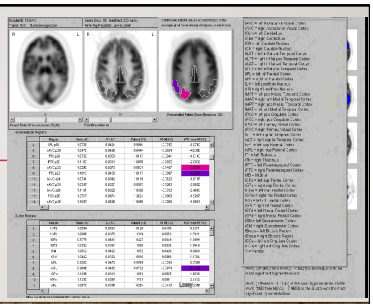
CASE 2

PET Scan



CASE 2

Neuro Q



PET REPORT

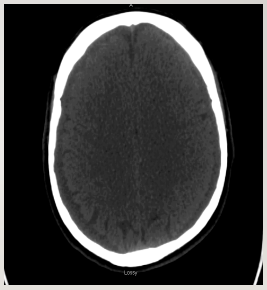
Report

- Abnormal study. Bilateral cortical hypometabolism in the parietal and posterior temporal lobes slightly greater on the right, and also involving the posterior Cingular cortex.
- This pattern is consistent with a neurodegenerative disorder and on balance, appearances favour an Alzheimer's type however Lewy bodies disease is a possibility.

CASE 2

CT Scan

Pretty Big Subdural!



CT REPORT

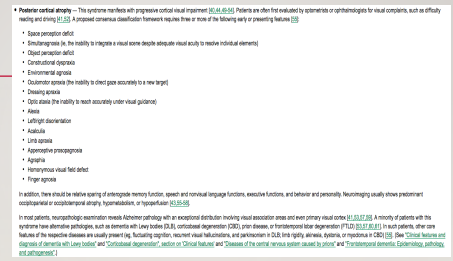
Report

- Limited CT images demonstrate bilateral low-attenuation collections within the convexities suspicious for subacute subdural haematomas or chronic hygromas. A dedicated CT brain is recommended to further evaluate. Correlation with any prior external imaging would be useful.

DIAGNOSIS

- Posterior Cortical Atrophy
- Variant of Alzheimers
- Typically complain of visual disturbance first, visit opticians etc.

POSTERIOR CORTICAL ATROPHY



- **Posterior cortical atrophy** – This syndrome manifests with progressive cortical visual impairment [M1,44,49-54]. Patients are often first evaluated by optometrists or ophthalmologists for visual complaints, such as difficulty reading and driving [51,52]. A proposed consensus classification framework requires three or more of the following early or presenting features [53]:
  - Space perception deficit
  - Simultanagnosia (ie, the inability to integrate a visual scene despite adequate visual acuity to resolve individual elements)
  - Object perception deficit
  - Constructional dyspraxia
  - Environmental agnosia
  - Oculomotor apraxia (the inability to direct gaze accurately to a new target)
  - Dressing apraxia
  - Optic ataxia (the inability to reach accurately under visual guidance)
  - Alexia
  - Left/right disorientation
  - Acalculia
  - Limb agnosia
  - Apperceptive prosopagnosia
  - Agraphia
  - Homonymous visual field defect
  - Finger agnosia

In addition, there should be relative sparing of anterograde memory function, speech and nonvisual language functions, executive functions, and behavior and personality. Neuroimaging usually shows predominant occipoparietal or occipitotemporal atrophy, hypometabolism, or hypoperfusion [M3,55-58].

In most patients, neuropathologic examination reveals Alzheimer pathology with an exceptional distribution involving visual association areas and even primary visual cortex [M1,53,57,59]. A minority of patients with this syndrome have alternative pathologies, such as dementia with Lewy bodies (DLB), corticobasal degeneration (CBD), prion disease, or frontotemporal lobar degeneration (FTLD) [55,57,60,61]. In such patients, other core features of the respective diseases are usually present (eg, fluctuating cognition, recurrent visual hallucinations, and parkinsonism in DLB; limb rigidity, akinesia, dystonia, or myoclonus in CBD) [59]. (See "Clinical features and diagnosis of dementia with Lewy bodies" and "Corticobasal degeneration", section on 'Clinical features' and "Diseases of the central nervous system caused by prions" and "Frontotemporal dementia: Epidemiology, pathology, and pathogenesis".)

CASE 2

- Met with patient and husband
- Disclosed diagnosis
- Commenced on Donepezil
- F/U in Dr Hutchinsons clinic
- F/U CT and MRI to ensure resolution of Subdurals
  - She was fine

CASE 3

- 62 year old gentleman
- PMhx: Depression, Anxiety, RTA significant head injury 1995 (full recovery, no neurological sequelae), migraine, HTN, weight loss (under investigation in SVUH)
- Former High Level Executive in HR in a Large Company
- Third Level Education

CASE 3

Subjective Hx:

- Feels he is becoming withdrawn
- Difficulty using technology
- Word-finding and naming difficulties
- Becoming more irritable and agitated

CASE 3

Collateral Hx:

- 2 year hx
- Word finding difficulties
- Telling fantastical/catastrophic stories
- ?Delusional beliefs/hallucinations
- Increased Impulsivity
- More Apathetic
- Reduction in Empathy
- Disinhibition
- Recent excessive drinking
- ?Aberrant motor behaviours

CASE 3

Collateral Hx

- Recent improvement in mood as anti-depressant increased
- Some safety concerns
  - Mistakes driving
  - Increased drinking
  - Leaving lights on, doors open etc

CASE 3

Exam

- Essentially normal physical exam
- Family gave him 20/27 on AQ
- Neuropsych testing:
  - Globally impaired, but not overwhelmingly so

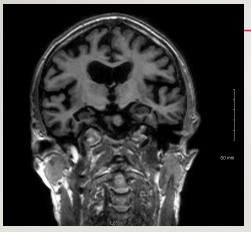
CASE 3

Neuroimaging

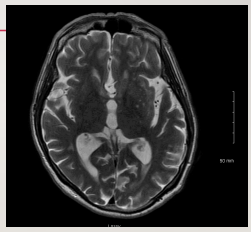
- Volume loss and ischaemic changes are more pronounced than you would expect for a patient of this age.
- Proportionate hippocampal atrophy on the background of generalised cerebral and cerebellar atrophy.

CASE 3

CORONAL



AXIAL

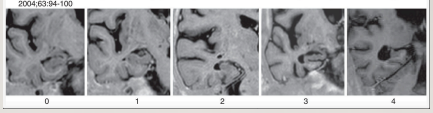


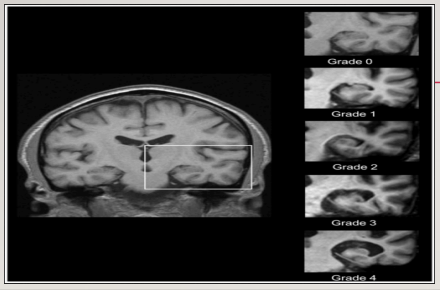
MEDIAL TEMPORAL LOBE ATROPHY

Medial temporal lobe atrophy

Score	Amplitude of the choroidal fissure	Amplitude of the temporal horn	Height of hippocampus
0	Normal	Normal	Normal
1	Slightly widened	Normal	Normal
2	Moderately widened	Slightly widened	Slightly reduced
3	Significantly widened	Moderately widened	Moderately reduced
4	Significantly widened	Significantly widened	Significantly reduced

Korf E. et. Al Medial temporal lobe atrophy on MRI predicts dementia with mild cognitive impairment. *Neurology* 2004;63:94-100

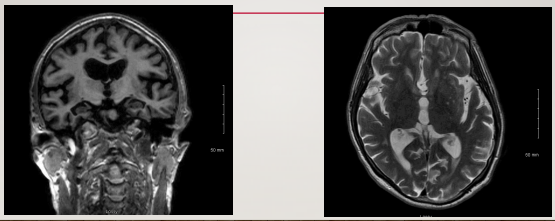




MEDIAL TEMPORAL LOBE ATROPHY

CASE 3

CORONAL AXIAL



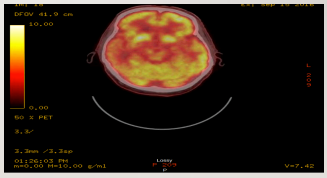
CASE 3

- Discussed at weekly case conference:
  - Definite issue here
  - Aetiology unclear, ?Atypical AD, ?FTD, ?C2H5OH
  - Memory impaired but not dramatically so
  - Some unusual behaviours emerging
    - Confounded by c2h5oh intake
    - Past psychiatric hx
- Plan
  - PET Scan
  - CSF Biomarkers

PET SCAN

REPORT PET SCAN

- Diminished metabolism confined to the right hemisphere.
- This may represent vascular pathology rather than a generalised neurodegeneration.



WHAT'S GOING ON?

RUN DMC – ITS TRICKY

NEXT STEP

- Discussed at Radiology Conference
  - Decision to perform New MRI
  - Wanted more sequences
  - In particular DWI and FLAIR
- CSF Still Awaited at this stage
- Repeat MRI
  - Background Diffuse Atrophy
  - Old Chronic Deep White Matter Ischaemic Changes
  - Nil Acute

CASE 3

- CSF was performed and results were awaited
- Met with patient and family again, ~4/12 after initial visit

CASE 3

Subjective Account

- Feels memory is worsening
- Losing things constantly
- Interview much more difficult
- Tangential
- Very confused/disoriented
- Pleasant, not aggressive

CASE 3

Collateral History

- Significant deterioration
- Pervasive anxiety
- Wandering around house
- Constant monologue about career and childhood
- Being looked after 24hrs a day
- No C2H5OH
- Abnormal jerky movements in his sleep
- +++sweet tooth
- Difficulty tying laces, leaving fly open
- Mis-identifying people
- Abnormal motor behaviour
- Wife summed up as "Just bizarre behaviour"

CASE 3

- Physical Exam
  - Essentially normal
  - Great Difficulty obeying commands during neuro exam
- Neuropsychological assessment
  - Deterioration across all domains
  - Camcog 65/107
  - Memory composite 17/27
  - MMSE Serial 7's 20, DLROW 22, Recall 1/3
  - RBANS – Immediate and delayed memory processing in the significantly impaired range

CASE 3

- CSF Biomarkers
- Normal Ranges
  - Total Tau: 135-345
  - Amyloid Beta: 591-997
  - Phosphorylated Tau: 35-64

General Immunology			
Total Tau	147.0		
Amyloid Beta 1-42	616.6		
Phosphorylated Tau	* 37.2		
Microbiology			
CSF Microscopy and Culture		CSF Microscopy and	

DIAGNOSIS

- Fronto-temporal Dementia
  - Behavioural Variant FTD
  - Other forms of FTD are variants of Primary Progressive Aphasia (nonfluent and semantic variants)
- bvFTD commonest FTD
- Age of onset typically 6th decade

DIAGNOSIS

- Disclosed diagnosis
- Put him off Road
- Discussed a will and enduring power of attorney (had been done at previous meeting)
- Informed of support groups
- Commenced on Trazadone for night time disturbance
- F/U Privately Dr Cunningham – Consultant Geriatrician in St James's
  - Under 65, issues with F/U for these patients depending on catchment area etc

BEHAVIOURAL VARIANT FTD

Clinical presentation — Early behavioral changes of bvFTD include the following:

- Disinhibition – Examples of disinhibition or socially inappropriate behavior include touching or kissing strangers, public urination, and flatulence without concern. Patients may make offensive remarks or invade others' personal space. Patients with FTD may exhibit utilization behaviors, such as playing with objects in their surroundings or taking others' personal items.
- Apathy and loss of empathy – Apathy manifests as losing interest and/or motivation for activities and social relationships. Patients may participate less in conversations and grow passive. Apathy is mistaken frequently for depression, and patients are often referred for psychiatric treatment early in the disease course.

  As patients lose empathy, caregivers may describe patients as cold or unfeeling towards others' emotions. Degeneration of right orbital frontal and anterior temporal regions may drive the loss of sympathy and empathy [5,6].
- Hyperorality – Hyperorality and dietary changes manifest as altered food preferences, such as carbohydrate cravings, particularly for sweet foods, and binge eating. Increased consumption of alcohol or tobacco may occur. Patients may eat beyond satiety or put excessive amounts of food in their mouths that cannot be chewed properly. They may attempt to consume inedible objects. This behavior correlates with right orbitofrontal, insular, striatal and hypothalamic degeneration [7-9].
- Compulsive behaviors – Perseverative, stereotyped, or compulsive ritualistic behaviors include stereotyped speech, simple repetitive movements, and complex ritualistic behaviors such as hoarding, checking, or cleaning. Other behaviors traditionally associated with obsessive compulsive disorder, such as hand-washing and germ phobias, are generally absent. Patients with FTD can develop a rigid personality, rigid food preferences, and inflexibility to changes in routine.

BEHAVIOURAL VARIANT FTD

- Typically do very well initially on neuropsych
  - See a change in the FrSBe (Factor analysis of the frontal systems behaviour scale)
- Normal neuro exam, maybe some frontal release signs e.g palmar grasp
- Imaging: Provides supportive but not diagnostic evidence
  - Fronto-temporal atrophy on MRI
  - Can see atrophy anywhere
- No treatment, no cure

DIAGNOSIS

- Clinical Diagnosis
  - Current 2011 International Behavioral Variant FTD Criteria Consortium based on Neary Criteria (1998)
- The FTDC criteria are structured as a diagnostic hierarchy. A diagnosis of possible bv

- FTD is based solely on the clinical syndrome and aims to identify patients at the mildest stages of disease. Possible bv FTD requires a combination of three of six clinical features:
  - Disinhibition
  - Apathy/inertia
  - Loss of sympathy/empathy
  - Perseverative/compulsive behaviors
  - Hyperorality
  - Dysexecutive neuropsychological profile
- A diagnosis of probable FTD is based on the same clinical criteria, plus demonstrable functional decline and imaging findings that reflect the principal anatomical location of neurodegeneration in bvFTD (ie, frontal and/or temporal lobe atrophy, hypometabolism, or hypoperfusion).

REFERENCES

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- https://www.mayoclinic.org/diseases-conditions/lewy-body-dementia/symptoms-causes/syc-20352025
- https://www.uptodate.com

QUESTIONS?