

I drink to forget....

- Alcoholic dementia
 - Overview
 - Case presentations
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 - Clinical Psychologist
 - Aged Care Services



Alcoholism cycle



DSM-V Substance-induced Major neurocognitive disorder

- Substance-Induced (Alcohol) Major Cognitive Disorder – includes both:
 - Korsakoff's Dementia
 - Core feature impaired memory
 - **Alcoholic Dementia** – *focus of this talk*
 - Core feature impaired executive functions

Sergei Korsakov (Korsakoff)

- PhD thesis on alcoholic paralysis
- Professor Extraordinarius - Moscow University Psychiatry Clinic
- Promoted rights of mentally ill
- Died of heart failure aged 46



WE/KS – aka “Wet brain” two syndromes, one cause

- Wernicke’s - reversible
 - Confusion
 - Ataxia
 - Nystagmus
 - EtoH withdrawal
 - If untreated may progress to KS

- Korsakoff’s- irreversible
 - Inability to form new memories
 - Severe memory loss
 - Confabulation
 - Hallucinations

Alcoholic Dementia – diagnostic criteria (Oslin, 1998)

- Dementia - persistent cognitive and functional decline following cessation of alcohol
- Significant alcohol use over an extended period
- Exclude other types of dementia
- ?Length and severity of alcohol use
- ?Minimum abstinence time

Alcoholic dementia

- A syndrome describing impaired executive function due to alcohol
- Frontal lobe problems
 - Impaired judgement
 - Difficulty making decisions
 - Lack of insight
 - Personality changes

Alcoholic dementia

- Unlike WKS, the brain damage is not primarily caused by thiamine deficiency
- Damage may be due to combination of both factors

Pathophysiology

- White matter loss in PFC, Corpus callosum, Cerebellum
- Gray matter loss in superior frontal association cortex, hypothalamus, cerebellum
- Altered glucose metabolism and perfusion in frontal lobes
- In WKS atrophy of the mamillary bodies, thalamus, cerebellum and frontal lobe

Is alcoholic dementia really just WKS in disguise?

- Epidemiological data suggest that ARD is more prevalent than WKS
- Need to demonstrate that direct neurotoxic effects of alcohol intake can lead to dementia over and above thiamine deficiency seen in WKS

Do the direct effects of alcohol exposure cause neuronal loss?

- Evidence
 - Animal studies suggest dose-related damage to memory and learning structures and disruption of neurotransmitters
- Imaging of alcoholics who don't have nutritional deficiencies or hepatic failure confirm these structural changes
- Unclear if these changes are permanent

Evidence for combined effect

- Interaction of Thiamine deficiency and alcohol neurotoxicity may increase white matter loss
- “Pure” cases of thiamine deficiency (without chronic excessive EtoH consumption) have a low rate of progression to KS

Beware: There is no standard “standard drink”

- The definition varies widely
- UK 8 grams
- Australia 10 grams
- USA 14 grams
- Japan 19.75 grams



How much alcohol must be consumed before damage occurs?

- 7-8 Australian std drinks/day (70-84 grams) over an extended period → • Cognitive “inefficiencies”
- >10 std drinks/day → • Cognitive deficits
- >49 (Men) std drinks/week → • Risk of Alcoholic dementia/WKS
- >40 (Women) over 5 years

Self-reported alcohol use

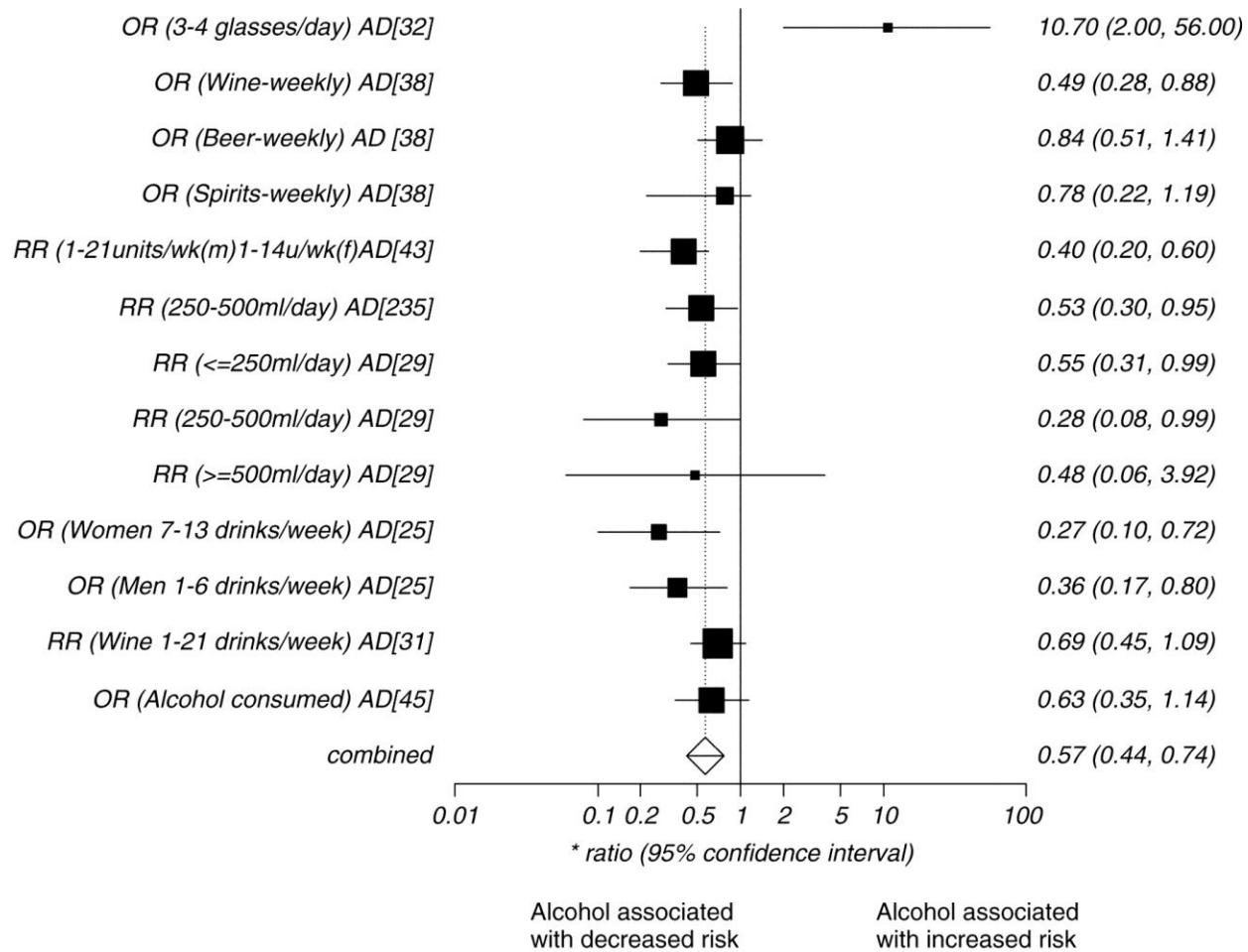
- Survey results of alcohol use account for approx 50% of reported alcohol sales (Chick & Kemppainen, 2007)
- Suggest that self-reported use may significantly underestimate actual use

Alcohol as risk factor for other forms of dementia

- Low levels of EtoH intake may reduce risk of dementia
- Peters et al (2008) meta analysis
 - Low EtoH may *protect* against AD
 - Risk ratio (RR 0.57; CI 0.44-0.74)
 - Not clear if this is true in women
 - Heavy use *increases* the risk
- In animal models low-dose ethanol prevents synaptic damage due to beta amyloid and α -synuclein

AD and alcohol.

Summary meta-analysis plot [random effects]



Ruth Peters et al. *Age Ageing* 2008;37:505-512

Recovery with abstinence?

- **Heavy alcohol consumption**
 - Deficits may resolve after 1 week of abstinence
 - Progressive recovery over 2 years
 - Visuospatial deficits slow to recover
 - Executive function, working memory and motor impairments may endure
- Pattern of binge drinking and withdrawal may exacerbate cognitive deficits
- Older age, female gender and low education is related to greater deficits and reduced rate of recovery

Recovery?

- **Korsakoffs**

- Damage regarded as permanent
 - But possible improvement in general knowledge, visual long-term memory, verbal fluency with 2 years of abstinence
- Victor et al (1971)
20% of KS patients can make of full recovery

- **Alcoholic dementia**

- Stability of cognition or possible improvement if abstinence maintained over 2 years

Neuropsychological profile for alcoholic dementia

- Impaired
 - Verbal learning and delayed recall (3 objects, retain an address)
 - Visuospatial ability (Clock drawing, Figure copying)
 - Executive functions, working memory (numbers backwards), motor speed, similarities, phonemic fluency
- Intact
 - Language skills
 - Alcoholic dementia > AlzD
 - Auditory recognition memory
 - Semantic fluency
 - Naming
 - General knowledge

Evidence base for test profile in Alcoholic dementia

- Caution
 - Few studies comparing ARD Vs Alz D
 - Small N studies
 - Heterogeneous p's
 - Uncertain neuropathology

Case 1

- 61 y.o. man
- Former Judge, now Barrister
- Ref to Memory Clinic
- STML - MMSE 28/30
 - Gradual onset over 18 months
 - Noticed by work colleague and wife
 - Patchy recall of conversations/events
 - Has to re-read legal docs
 - No longer taking on complex cases

Medical history

- Hypertension
 - Hyperlipidaemia
 - GORD
 - Gout
 - Iga nephropathy
-
- MRI (2012) shows age-related atrophy and svd

Medical history

- No personal hx of stroke
- Concussion x 2 as young adult
- Family hx
 - Mother “forgetful” post-surgery 80 yrs
 - Father d. aged 70’s but cognitively intact
- No psychiatric hx
- Smoking – Ceased 1990 < 1 pack/day
- EtoH – 40g/day long term
 - Reduced 4/12 ago to odd glass of wine

Clinical observations

- Discursive during interview
 - Mildly disinhibited during testing
 - Socially appropriate
 - Retained insight
-
- Normal range for mood sx in previous wk

Neuropsych test results

Domain		Description	Percentile
Premorbid IQ		Very Superior	>98%
Orientation		Normal	
Attention		Average	50%
Auditory recall	Immediate	Average	50%
	<i>Delayed</i>	<i>Impaired</i>	<i><5%</i>
	Recognition	Intact	
Visual recall	Immediate	Low avg- Average	25-50%
	<i>Delayed</i>	<i>Impaired</i>	<i><5%</i>
	Recognition	Intact	

Neuropsych test results

Domain		Description	Percentile
Language	Naming	Normal	
	Similarities	Very Superior	>98%
	Word meanings	Very Superior	>98%
Visuospatial	Figure copy	Intact	
	Block design	Very Superior	>98%
Executive functions	Cognitive flexibility	High Average	75%
	Inhibition	Average	50%
	Sorting	Superior	90%
Verbal fluency	Phonemic	Superior	90%
	Semantic	Average	50%

Diagnosis

- MCI – Amnestic
- Role of EtoH?
- Intake high but
 - Orientation, motor speed, visuospatial intact
 - Language intact in Alcoholic dementia but here semantic fluency < phonemic (seen in early AD)
 - Benefit from recog cues suggests retrieval problem (seen in AlcD but also VaD)

Case 2

- 70 y.o. woman
- Former Publicist/PA
- Separated, lives alone in unit
- Ref by GP for cognitive ax
- Also ref for ACAT ax
- Long hx of EtoH abuse
- MMSE 25/30

Medical history

- IBS
- Polymyalgia rheumatica
- Laminectomy, Discectomy

- Multiple ED admissions (2010-2014)
 - Falls, Fractures (elbow, shoulder, radius, ribs), Facial lacerations, Haematoma
 - Measured BAC's 0.22-0.36

Medical history

- CT Brain (multiple)
 - No acute injury
- MRI in 2014
 - Moderate degree of generalised atrophy and mild to moderate svd

Psychiatric history

- Anxiety/depression
- First s/b Psychiatrist in 1990-91
 - Extreme distress, suicidality
 - Rx ADT
 - Problems resolved when she found a new job
- Medical records file notes suggest problems coping with adverse life events
 - Depression and alcohol abuse noted when lost job, sister died, hb left

Involuntary admissions

- August 2013 scheduled by GP under MHA – 6 day adm to RNSH 9E
- Did not engage with D&A services following discharge
- Dec 2014 found at home intoxicated and doubly incontinent. Very poor personal hygiene. Conditions inside the unit described by Ambulance service as the worst they have seen
- Scheduled by GP – adm to RNSH – Riverglen – Northside clinic

Post-discharge

- Drinking 1-2 bottles wine most days
- Not taking thiamine, ADT
- Disoriented to time & place
 - Calling concierge after hours to let her in
 - Asking strangers to help her to get home
- Estranged from family
- Unit returning to state of squalor

Corroborative hx

- Both sons, d-i-l, ex-hb, and sister all able to verify hx
- Son states that
 - Bills not being paid
 - Divorce papers left aside
- Efforts by all family to assist futile
- Ceased contact with sister (prev v.close)

Neuropsych test results

Domain		Description	Percentile
Premorbid IQ		Average	50%
Orientation		Impaired	8/14 correct
Attention		Average	50%
Auditory recall	Immediate	Impaired	<5%
	Delayed	Impaired	<5%
	Recognition	Impaired	
Visual recall	Immediate	Impaired	<5%
	Delayed	Impaired	<5%
	Recognition	Low Average	(2/7 correct)

Neuropsych test results

Domain		Description	Percentile
Language	Naming	Low Average	<25%
	Colour naming	Average	50%
	Word reading	Low Average	<25%
Visuospatial	Block design	Borderline	<9%
Executive functions	Cognitive flexibility	Impaired	<1%
	Inhibition	Impaired	<1%
	Switching	Impaired	<1%
Verbal fluency	Phonemic	Borderline	<9%
	Semantic	Impaired	<5%

Diagnosis

- Probable Alcohol-related dementia
 - With ?cont from cerebrovascular disease
- Probably not Alz D/FTD

- But with the caveat that patient is still drinking so criteria for persistent deficits following cessation not demonstrated

Recommendations

- Family conference
 - Application to NCAT (Guardianship)
 - Appt of Financial Manager (son)
 - Risk mgmt plan
 - Medical review ?adm detox adm prior
- Placement in RACF
 - Medications, nutrition, hygiene, abstinence, social stimulation

Summary

- DSM-V Substance-Induced (Alcohol) Major Cognitive Disorder – includes both
 - WKS and Alcoholic dementia
 - Korsakoffs primarily affects memory
 - Alcoholic dementia affects executive functions
- The latter is a combination of neurotoxic effects of excessive alcohol and thiamine deficiency over long-term
- Cognitive and functional deficits persist following cessation
- Need to exclude other forms of dementia

Questions/Comments

