Psychosis and Acquired Brain Injury

When Two Worlds Collide

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A few stats!

• 1 million people each year attend A &E dept with a brain injury

• 150,000 mild

• 10,000 moderate

• 11,000 severe
Demographics

- Peaks at below 5 years, 15-24 yrs, +70 yrs
- Males are two times more likely than females to sustain a brain injury.
- The highest rate of injury is for males age 15-24.
- Previous TBI
  - After 1st TBI, risk of second injury is 3 times greater
  - After 2nd, risk of third injury is 8 times greater
- Drug / Alcohol misuse and dependency
- Lower SE status / unemployed / forensic history
- Mental illness
Shared demographics with schizophrenia
ICD-10 codes

- Delirium F05
- Organic hallucinosis F06.0
- Organic catatonic disorder F06.1
- Organic delusional disorder F06.2*
- Organic personality disorder and schizophrenia F07.0 / F20 *
- Other specified mental disorder due to brain damage and dysfunction – Epileptic psychosis NOS F06.8
ABI and psychosis:

- Immediate – delirium
- Post-ABI delayed psychosis
- Post and inter-ictal psychosis
- Comorbidity – schizophrenia / schizoaffective / bipolar
Delirium

- Early clinical features of delirium: confusion, inattention, cerebral disorganization and psychotic symptoms – hallucinations can be prominent.

- When the patient emerges from delirium, more discrete psychotic features can develop - characteristic symptoms include delusional disorientation, delusional misidentification and confabulation. Confabulations may become chronic.

- Management – identify cause (withdrawal/intoxication), avoid polypharmacy, maintain safety, replace electrolytes/nutrition. Low dose haloperidol/ risperidone/ benzodiazepines (with caution)

- High mortality rates and increased association between delirium and later development of psychosis.
The onset is often gradual, with a subacute or chronic course (Zhang & Sachdev, 2003).

Variable data - ~1.5 % with ABI develop psychosis. Increases over time (up to 10%)

Prodrome – bizarre behaviour (50%) depression and affective instability(39%) antisocial and inappropriate social behaviour (36%), social withdrawal (31%) and deterioration at work (33%)

Time of onset -  Sachdev and colleagues (2001) reported a mean latency of 54.7 months between head injury and onset of psychosis, Other studies have shown bimodal distribution<1 year and 4-5 years post ABI. Reported cases up to 20 years latency.
Variable data regards location and type (ie open / closed) of brain injury.

Most significant risk factors are:-

- Family history of schizophrenia
- Early age at time of ABI
- male
- Severity of injury – moderate to severe (can occur following mild ABI esp if repeated)
- premorbid psychiatric illness – diagnosis of schizophrenia x 2 more likely than general population to have a brain injury
- Neurological / developmental disorder
- Epilepsy
Clinical features

- Persecutory delusions
- Auditory hallucinations
- Hallucinations associated with neurological deficits
- Misidentification/ misinterpretation of normal stimuli of normal stimuli – Capgras, Fregoli
- Uncommon thought disorder, loosening of associations
- May have retained insight
- Fewer negative symptoms
- Cognitive impairment – memory, executive function impairment (94%)
- MRI/EEG findings
- Epilepsy - ~30%
Cognitive impairment

• Memory impairment – recent memory and processing speed

• Impairment of executive functioning (frontal lobe syndrome)
  • Planning
  • Organization
  • Ability to adapt emotions or responses
  • Impulsivity/ disinhibition
  • Apathy - Apathy alone - prevalence 10% – disinterest, disengagement, inertia, lack of motivation, lack of emotional responsivity

• Changes to gait and speech (not usually formal thought disorder)
# Investigations (Fujii 2012)

<table>
<thead>
<tr>
<th></th>
<th>PD-TBI</th>
<th>Schizophrenia</th>
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<tbody>
<tr>
<td><strong>Presence of Negative Symptoms</strong></td>
<td><strong>37%</strong></td>
<td><strong>50%–90%</strong></td>
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<td></td>
<td><strong>64% blunted affect</strong></td>
<td><strong>46% social withdrawal</strong></td>
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<tr>
<td></td>
<td><strong>45% social withdrawal</strong></td>
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<tr>
<td><strong>MRI/CT: Positive Findings</strong></td>
<td><strong>70%</strong></td>
<td><strong>12%–35%</strong></td>
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<tr>
<td>Atrophy</td>
<td><strong>6%</strong></td>
<td><strong>12%–35%</strong></td>
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<tr>
<td>Focal abnormalities</td>
<td><strong>100%</strong></td>
<td><strong>6%–9%</strong></td>
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<tr>
<td><strong>Most common finding</strong></td>
<td><strong>74% frontal</strong></td>
<td><strong>Whole brain</strong></td>
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<td></td>
<td><strong>47% temporal</strong></td>
<td>Hippocampal atrophy</td>
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<td><strong>21% enlarged ventricles</strong></td>
<td>Enlarged ventricles</td>
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<td><strong>SPECT/PET</strong></td>
<td><strong>46% temporal</strong></td>
<td>Hypofrontality</td>
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<td></td>
<td><strong>38% frontal</strong></td>
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<tr>
<td><strong>EEG: Positive Findings</strong></td>
<td><strong>77%</strong></td>
<td><strong>20%–60%</strong></td>
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<tr>
<td>Most common finding</td>
<td>Temporal spiking/slowing</td>
<td>Slowing (delta)</td>
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<td>Presence of Negative Symptoms</td>
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<td><strong>Most common finding</strong></td>
<td><strong>42% frontal</strong></td>
<td><strong>22%–35%</strong></td>
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<td><strong>27% temporal</strong></td>
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Ictal psychosis – complex partial and absence status can mimic psychosis

Postictal events

- Criteria proposed by Stagno for postictal psychosis include the following:
  - Psychotic symptoms occur after a seizure or, more frequently, a series of seizures, after a lucid interval, or within 7 days of the seizure(s)
  - The event is time-limited, lasting days or, rarely, weeks; no significant clouding of consciousness occurs
- Often delusions and hallucinations are well systematised

Inter-ictal psychosis

- unremitting chronic interictal psychosis seen in long-standing epilepsy
Does ABI cause Schizophrenia?

- Significant association between Schizophrenia and ABI (odds ratio 1.65%) 
  Molloy, Conroy et al Schiz bull 2011;37 (6) 1104-1110

- Early onset psychosis more likely to have had a ABI 
  Abdel Malik et al 2003

- Largest effect is if genetic predisposition and ABI

- Confounding factors ie drug misuse
When the 2 worlds collide, how do you decide?

• Symptoms don’t quite fit
• Poor compliance/ disengagement
• Frequent outbursts / low frustration tolerance
• Treatment resistance
• Deterioration despite treatment
Assessing ABI

- History – ”ABI checklist for psychiatrists”  
  (Simon Fleminger – SoN website)
- ACE-R – comprehensive cognitive assessment
- Neurological examination
- Neuropsychology assessment
- OT, SALT and physio
- Other tests – MRI (not CT) and EEG
Epilepsy

Diabetes

Mental illness
• Depression
• Anxiety
• Sleep disorders

Substance misuse/withdrawals

Pain

Treat Co-morbidity
Get Environment Right

- Space
- Noise
- Structure
- Temperature
- Consistency
• Start low, go slow”
• Some evidence to suggest that dopamine antagonists can impede brain recovery Feeney et al 1982
• Try to avoid benzodiazepines – worsen cognitive functioning
• Mindful of seizure threshold
• Lower threshold for sedation
• Increased vulnerability to EPSE and anticholinergic effects
• Case reports/ anecdotes – risperidone, olanzapine, quetiapine, haloperidol (dose dep), ??aripiprazole. Every brain injury is different!
But...what are we treating?

Pharmacological management for agitation and aggression in people with acquired brain injury
Simon Fleminger, Richard RJ Greenwood, Donna L Oliver (Cochrane review 2008)

“This review found no firm evidence that drug management of agitation and aggression in adults with acquired brain injury is effective.

There was weak evidence, based on a few small randomized controlled trials, that beta blockers can improve aggression after acquired brain injury, but very large doses were used which would have been likely to produce significant adverse effects. For other classes of medication, reasonable size randomized controlled trials have not been published.

Based on the lack of evidence, the review comes to no conclusion on the effectiveness of drugs. There is reasonable anecdotal evidence, for example in published cases series, that antipsychotics, mood stabilizers and antidepressants may be effective in the management of this situation.”
Cons

- Seizure threshold
- Sedative effects
- Increase adverse effects Michals et al Journal of clinical Psychopharmacology 1993
- Treatment resistance – assumes correct diagnosis

Pros

- Effective in stabilizing psychosis
- Reduction in violent aggression John E Kraus et al Journal of Neuropsychiatry and Clinical Neurosciences 2005
- Enforced monitoring
Clarity of diagnosis

Dose effect

If epilepsy history consult with neurologist. EEG monitoring and antiepileptic dose adjustment

If no history of epilepsy but ABI very careful monitoring of levels. If severe ABI or hypoxic injury consider prophylactic antiepileptic if unsure consult neurologist and peer review.
Resources

- RCPsych – Faculty of Neuropsychiatry
- British Neuropsychiatry Association
- Brain Injury Social Workers Group (BISWiG)
- Headway UK
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