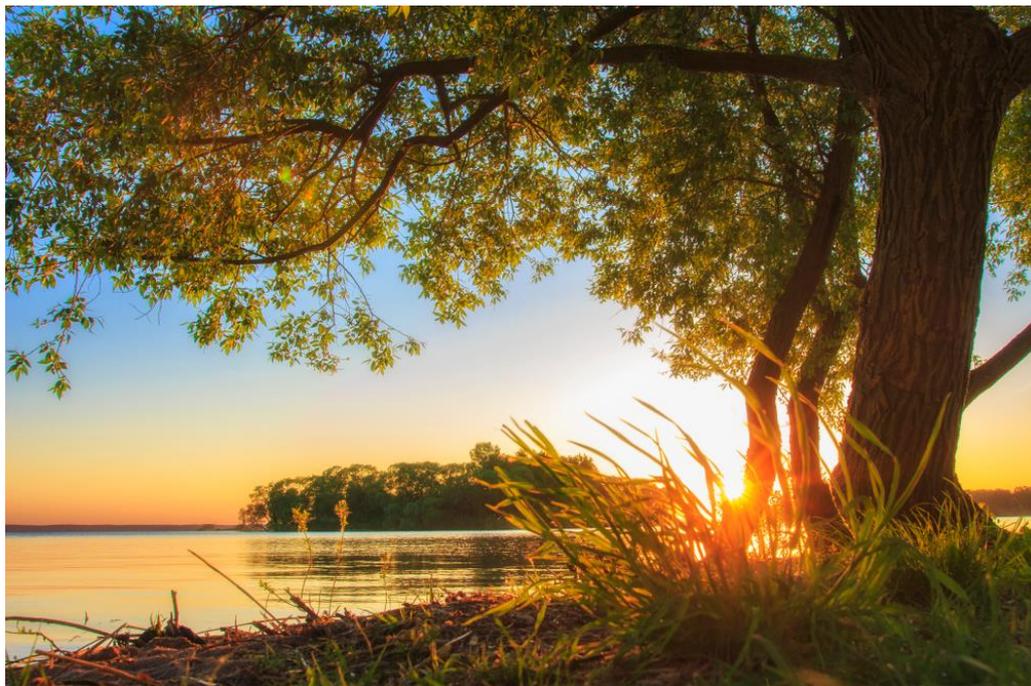


Summer 2020



Neuropsychiatry Newsletter

The voice of the Faculty of Neuropsychiatry



Featuring

- Highlights from the Faculty of Neuropsychiatry Annual Conference
- Wilsons Disease: A Review
- Latest News from the Executive Committee

Neuropsychiatry Newsletter

Summer 2020
Edition

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Editorial

On Covid-19 and its aftermath

by

Dr Kevin Foy

Editor of Neuropsychiatry Newsletter

Consultant Neuropsychiatrist, Dublin

Brexit, Liberty Protection Safeguards and the Australian Bush fires at this stage all feel like the problems of a different Age. As Yeats said about a different period of tumult, *"all is changed, changed utterly, a terrible beauty is born"*. From this standpoint, the magnitude of those changes are still unclear as indeed is whether the pandemic is truly under control. The spectre of second and further waves are still possible. Similarly, the potential of protection from any of the vaccines in development remains unclear. Regardless of these uncertainties, what is certain is that the world post covid-19 will be a different one. Medicine will have changed. Neuropsychiatry will have changed. We will have changed.

During the dark uncertain days of the crisis, incoming chair of the faculty of neuropsychiatry, Mike Dilly, spearheaded plans for neuropsychiatry at a national level and describes those plans in this edition of the newsletter.

Dot Bindman provides an update on the latest developments from the executive committee of the faculty.

Metabolic conditions such as Wilson's disease can create a lot of confusion. Samuel Shribman and Professor Eileen Joyce provide a nice summary of the key facts about this under diagnosed condition. Professor Hugh Rikards also writes an opinion piece on the ongoing exclusion of people with neurological conditions from mainstream psychiatry.

We look back at those apparently simpler days of the most recent annual meeting of the faculty last September with articles from the prize winning research.

In conclusion, and on behalf of the faculty, I hope that you and yours have managed to stay well and perhaps even flourished during this difficult epoch.

News from the Executive Committee

This year's strategy meeting, held remotely in May, was particularly full as it marked the end of Professor Joyce's tenure as committee chair and the inauguration of our new chair, Dr Mike Dilley.

Before moving to the main agenda items of discussing past and future strategic aims, there are two important pieces of news. Firstly, in line with the college's policy on postponing all face-to-face meetings until January 2021, this year's annual Neuropsychiatry Faculty conference in September will be held remotely on 18 September. Further details can be found on the final page of the newsletter or on www.bit.ly/Neuro20. Also highlighted was the work of committee member Tim Nicholson and colleagues on the Coronerve surveillance programme <https://www.rcpsych.ac.uk/members/your-faculties/neuropsychiatry/coronerve-surveillance-survey>. This national collaboration between neurology and psychiatry has already collected and published findings from a preliminary dataset and clinicians are encouraged to continue reporting cases via the weblink or through the Covid pages on the College website.

Reviewing the strategic targets set when Professor Joyce became Chair four years ago, the committee paid tribute to her outstanding leadership. Significant progress has been made towards a Neuropsychiatry curriculum, which is being overseen by the College. Higher Learning Outcomes were submitted for approval in February this year. Professor Joyce will continue this work for the College, with the next stage being submission of key capabilities. She will also further develop the integration of capabilities in Neuropsychiatry into Neurology training through collaboration with the Association of British Neurologists.

Work by a number of committee members on commissioning has come to fruition in the last year with the approval by NHS England of a new speciality Neuroscience specification including Neuropsychiatry. This mandates the commissioning of Neuropsychiatry in every neuroscience centre in England.

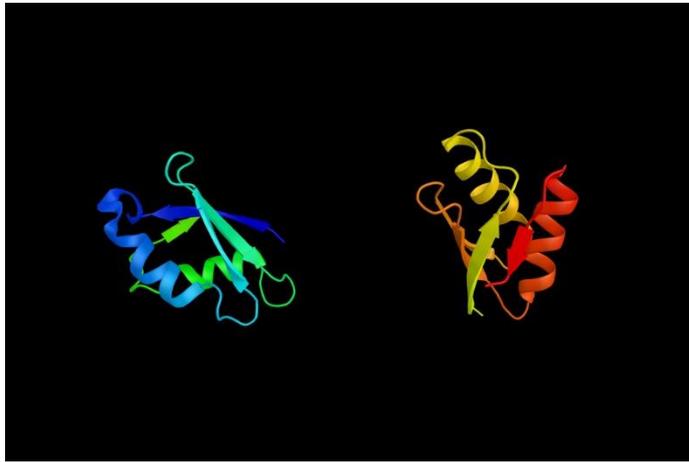
Providing educational meetings of a high standard to promote the clinical neurosciences relevant to the practice of psychiatry was the final aim set in 2016. The faculty conference has increased in popularity, with the last 3 years oversubscribed. Collaboration with the International Neuropsychiatric Association on this year's conference will continue the

tradition of talks from internationally renowned speakers and bring an even wider audience from around the world. The faculty have also had success closer to home in staging regional events, including well-attended meetings in Cardiff and Belfast.

The strategy day closed by welcoming new Chair, Mike Dilley, who introduced the strategy for the next four years. In the forthcoming autumn edition of the newsletter, Dr Dilley has kindly agreed to set out his vision for the faculty and give an insight into his work of adapting regional neuropsychiatry services to cope with the coronavirus pandemic.

Clinical presentations and diagnostic challenges in Wilson's disease

Samuel Shribman, Thomas T Warner, Eileen Joyce



Wilson's disease (WD) is a rare autosomal-recessive disorder of copper metabolism caused by mutations in the *ATP7B* gene that presents with a range of hepatic, neurological and psychiatric manifestations.¹ It often mimics common conditions and, in combination with the lack of any single reliable diagnostic test, poses a significant diagnostic challenge to neurologists and psychiatrists; the average delay from symptom onset to diagnosis is two years.

In parallel, WD is one of the few neurodegenerative disorders for which disease-modifying treatments (chelation therapy) are highly effective at reducing and preventing morbidity. Delayed or missed diagnoses can therefore lead to avoidable neurological and psychiatric disability. Here we discuss the clinical presentation and the investigation and diagnosis of WD to aid the practising psychiatrist.

Clinical presentations

The majority of patients present between age 5 and 35 years; however, presentations have been reported up to 72 years.² The psychiatrist may be the first specialist to encounter the patient but some patients may have been misdiagnosed with alternative neurological or hepatic diseases by other specialists. The combination of a psychiatric presentation with any liver disease or a movement disorder, particularly in a children or young adults, should raise suspicion of WD.

Hepatic presentations can be acute or chronic ranging from incidental abnormal liver function tests to cirrhosis or acute liver failure. While

deranged liver functions tests would clearly prompt a physician to consider liver disease, a low platelet count may be the only manifestation of cirrhosis in some cases. Some patients also develop a haemolytic anaemia manifesting with recurrent episodes of jaundices and prompting investigation by haematologists.

Neurological presentations are classically with a subacute or chronic movement disorder. However, bulbar symptoms, such as dysarthria or drooling, occurs in 90% of these cases. The movement disorder is usually characterised by tremor, dystonia or parkinsonism or a combination. After speech disturbance, postural tremor of the upper limbs is the most common neurological examination finding. Other neurological features such as ataxia, chorea, spasticity and seizures can occur and executive dysfunction, which may lead to difficulties at school or work, is common. Importantly, the patient may present with one movement disorder, such as a tremor, but develop additional features, such as speech or gait disturbance, over the following months.

Ophthalmological presentations are rare but copper deposits in the cornea, Kayser-Fleischer (KF) rings, can be detected in most patients. These may be visible at the bedside as brown or yellow discoloration at the periphery of the cornea. Slit-lamp examination by an experienced ophthalmologist is required to confirm their presence and WD should not be excluded on the absence of KF rings.

Psychiatric symptoms precede the movement disorder in 20% and are present in 30-40% by the time of diagnosis. Overall, 66% develop psychiatric symptoms at some point during the illness. Wilson's disease has been referred to as the 'great masquerador' because it can present with symptoms common to most psychiatric disorders. In childhood the most common feature is personality change and decline in school performance. However, psychosis, tics, OCD symptoms and even catatonia have also been documented. In adults, more than 50% present with personality change characterised by irritability, aggression, disinhibition and impulsivity, emotional lability and paranoia. Symptoms of mood disorders are also recognised with 4% meeting criteria for major depressive disorder and 14-18% for bipolar disorder. Of the psychosis symptom spectrum, paranoid delusions are most common but visual and auditory hallucinations have been documented. Finally, 25% patients with Wilson's disease show cognitive decline; this can be of a dysexecutive syndrome, affecting working memory and processing speed, or a more rarely global impairment akin to dementia.

Investigation and diagnosis

This can be straight-forward or challenging. Serum caeruloplasmin represents a simple screen for WD that can be performed with a random blood test. However, the sensitivity and specificity varies depending on the presentation and it cannot exclude WD. While there is limited data on

isolated psychiatric presentations (without overt neurological or hepatic features), around 85% of neurological and 59% of hepatic presentations have a serum caeruloplasmin less than 0.2 g/L. Typical neurological and psychiatric symptoms with a caeruloplasmin below 0.1 g/L makes the diagnosis highly likely.³ Counter-intuitively, the total serum copper is typically low in WD given the majority of circulating copper is bound to caeruloplasmin.

Urine copper output measured over a 24-hour collection are over 1.6 μmol in 78% and KF rings are detected by an ophthalmologist in 90% of neurological presentations. Genotyping of the ATP7B using exome sequencing detects two mutations in 98% of cases but takes several months at least, during which time the patient is likely to deteriorate. Liver biopsy is another diagnostic tool but is clearly invasive.

An MRI of the brain using standard T1- and T2-weighted sequences is abnormal in 99% of patients with neurological patients. The typical abnormalities are bilateral T2-weighted hyperintensities in the basal ganglia, thalamus and brainstem. When mild these can be dismissed as 'non-specific' and when focussed in the brainstem might be misdiagnosed as inflammatory lesions of multiple sclerosis. Mild atrophy is not uncommon at presentation and some patients, particularly those with prominent liver disease, may have T1-weighted hyperintensity in the basal ganglia. The sensitivity of MRI for isolated psychiatric presentations is unclear but abnormalities are not uncommon in hepatic presentations without neurological symptoms.

In the neurology clinic, we send a serum caeruloplasmin and copper for any patient presenting with a movement disorder (tremor, dystonia, parkinsonism, chorea and ataxia) where we have a low suspicion of WD. Many of these patients will have an MRI, which if completely normal, is also reassuring. We do not screen patients with isolated cervical dystonia or those presenting with asymmetrical parkinsonism after the age of 50 years in whom WD is very unlikely. We arrange a full battery of testing with serum caeruloplasmin, serum copper, 24-hour urine collection, MRI brain and ophthalmology referral for slit lamp in those patients we consider to have red flags. This includes those with a subacute onset, bulbar involvement, concomitant liver or psychiatric disease, a family history or suspicion of KF ring. Biochemical test results can take several weeks and so serum and urine tests should be arranged immediately and patients should be advised to make contact if any deterioration in the interim. The Leipzig criteria can be used to confirm the diagnosis of WD where initial biochemical testing is inconclusive.⁴

For those patients initially presenting to mental health services, the approach is less clear. Given the diverse range of psychiatric manifestations screening all referrals with serum caeruloplasmin is not feasible. We advocate urgent referral to a movement disorder specialist neurologist or a hepatologist in the presence of any red flags. Arranging a full blood count,

liver function tests and serum caeruloplasmin in such patients is relatively quick and cheap and may help reach a prompt diagnosis in some cases. If further specialist opinion is delayed then a full screen of diagnostic tests may need to be initiated by the psychiatrist.

In those patients with a known liver disease or movement disorder, the possibility of WD may not have previously been considered and the onus is therefore on the psychiatrist to refer to a specialist for diagnostic testing. Children and young adults with psychiatric presentations later develop a suspected drug-induced movement disorder pose a particular challenge and the possibility of WD requires careful consideration.

Treatment of neuropsychiatric symptoms

If caught early enough, neuropsychiatric symptoms can improve with the primary treatment of Wilson's disease aimed at removal of copper deposition. Standard psychotropic medication is often effective for specific symptoms but the choice is limited if there is liver involvement, e.g. sodium valproate, phenelzine, imipramine, amitriptyline, duloxetine, bupropion, agomelatine are relatively contraindicated, and some antipsychotics worsen movement disorders, e.g. haloperidol and risperidone. For mood stabilisation lithium is preferred as it is not metabolised by the liver but carbamazepine and lamotrigine are alternatives if lithium worsens tremor. Olanzapine, quetiapine and clozapine are effective for psychosis, as are SSRIs for depression. ECT has successfully treated severe depression and catatonia in people with Wilson's disease.

Summary

Though a rare condition, Wilson's disease is a potentially reversible cause of neurological, psychiatric and hepatic symptoms. Diagnostic testing for it is simple and cheap. Clinicians need to be aware of it as a potential diagnosis in patients presenting with psychiatric symptoms with movement disorder or hepatic symptoms.

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Dr Samuel Shribman is a clinical research fellow and neurology trainee at the UCL Queen Square Institute of Neurology. He works in the multi-disciplinary Wilson's clinic and runs a longitudinal, observational study on Wilson's disease at the National Hospital for Neurology and Neurosurgery.

Professor Thomas Warner is director of the Queen Square Brain Bank and Reta Lila Weston Institute for Neurological Studies. He is a movement disorders specialist at the National Hospital for Neurology and Neurosurgery and his research focusses on the molecular pathogenesis of a number of hereditary movement disorders with the aim of developing novel translational strategies.

Professor Eileen Joyce is professor of neuropsychiatry at the National Hospital for Neurology and Neurosurgery and UCL Queen Square Institute of Neurology. Her special interest is in the neuropsychiatry of movement disorder. She is also chair of the executive committee of the faculty of neuropsychiatry.

Faculty of Neuropsychiatry Annual Conference Winners

Cognitive Behavioural Therapy for Dissociative Seizures and Co-existing Epilepsy: Outcomes from a Tertiary Neuropsychiatry Service

Alex Berry

Though figures vary widely, it's estimated that approximately 12% of those with epilepsy also experience dissociative seizures (also known as functional seizures or psychogenic non-epileptic attacks); and that 22% of those with dissociative seizures have epilepsy ¹. Those with dissociative seizures co-existing with epilepsy represent a relatively under-researched population. To put this into some perspective, the 4th Edition of Lishman's Organic Psychiatry contains an 88-page chapter on epilepsy, yet only 1 paragraph within this chapter specifically addresses those who have epilepsy with co-existing dissociative seizures ².

Patients with co-existing dissociative seizures and epilepsy tend to show a consistent pattern of epilepsy diagnosis preceding the onset of dissociative seizures, often by many years ³. Some semiological features of dissociative seizures are less frequently observed in those with dual diagnosis, such as opisthotonic posturing. In contrast "total lack of responsiveness" and myoclonic seizure semiology are more frequently observed amongst those with dual-diagnoses compared to those with uncomplicated dissociative seizures. The CODES trial is the first multi-centre, adequately-powered randomised controlled trial for CBT in those with dissociative seizures, and the results of which are currently awaiting publication. Notably, the CODES trial did not include patients with active epilepsy.

At the National Hospital for Neurology and Neurosurgery (NHNN), patients can be referred for a 12-session course of outpatient cognitive behavioural therapy (CBT) as treatment for dissociative seizures. Whilst there is some evidence to support the use of CBT-based therapy treating the psychosocial aspects of epilepsy, it's possible that the presence of co-existing epilepsy may complicate delivery or reduce effectiveness of CBT for dissociative seizures ⁴.

Methods

We designed a simple service evaluation project investigating clinical outcomes, demographic details and dissociative seizure

semiology amongst those with dissociative seizures and co-existing epilepsy who underwent a course of CBT for dissociative seizures at the NHNN. We performed a retrospective case note review of all patients who underwent CBT at the NHNN neuropsychiatry department over a two year period, identifying 170 cases, of which 13 (8%) were patients who had dissociative seizures with co-existing epilepsy.

Results

In this dual diagnosis group, mean seizure frequency reduced to almost 50% by the last session of CBT, though this didn't reach statistical significance. All patients undertook 3 rating scales at the first and final sessions of therapy: the Patient Health Questionnaire-9 (PHQ-9), Generalised Anxiety Disorder-7 (GAD-7) and the Work and Social Adjustment Scale (WSAS). Significant reductions were observed in PHQ-9 and GAD-7 scores at the end of therapy, and a non-significant reduction was observed in the WSAS score.

Epilepsy diagnosis preceded diagnosis of dissociative seizures in all cases, with a mean interval of 22.5 years (range 2-48.5) between epilepsy-onset and dissociative seizure-onset.

Psychiatric comorbidity was found in 12 patients (92.3%), mainly consisting of depression and anxiety. Adverse life events occurring before the age of 18 were documented in 9 patients (75%).

Possible precipitating events for dissociative seizures were identified in 9 (75%) patients – with school or work stressors identified in 5 (38.5%), and intramarital/relational difficulties in 2 (15%). Childbirth, post-epilepsy surgery, bereavement and acute physical illness were identified as potential precipitants in individual patients.

"Drop-attack" type episodes and post-ictal states were described in 5 patients (39%), whilst opisthotonic posturing and generalised convulsions were relatively infrequently identified during dissociative seizures (2 patients).

Conclusion

Whilst this work is part of a preliminary service evaluation, the findings appear broadly in line with what has currently been published in the literature about this important patient group, and it is encouraging to see that there may be some benefits to using CBT

in these dual-diagnosis patients. More work is clearly needed in evaluating treatment options for dissociative seizures, and in increasing recognition of those with both epilepsy and coexisting dissociative seizures.

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Dr Alex Berry is an ST4 in general adult psychiatry in North Central London. He graduated from the Brighton and Sussex Medical School, and completed an intercalated BSc in Neuroscience at Sussex University. His foundation years included posts in both neurology and psychiatry. He completed core psychiatric training in North Central London, which included a placement in neuropsychiatry at the National Hospital for Neurology and Neurosurgery, Queen Square.

Do traumatic brain injuries increase the risk of developing mental health disorders later in life?

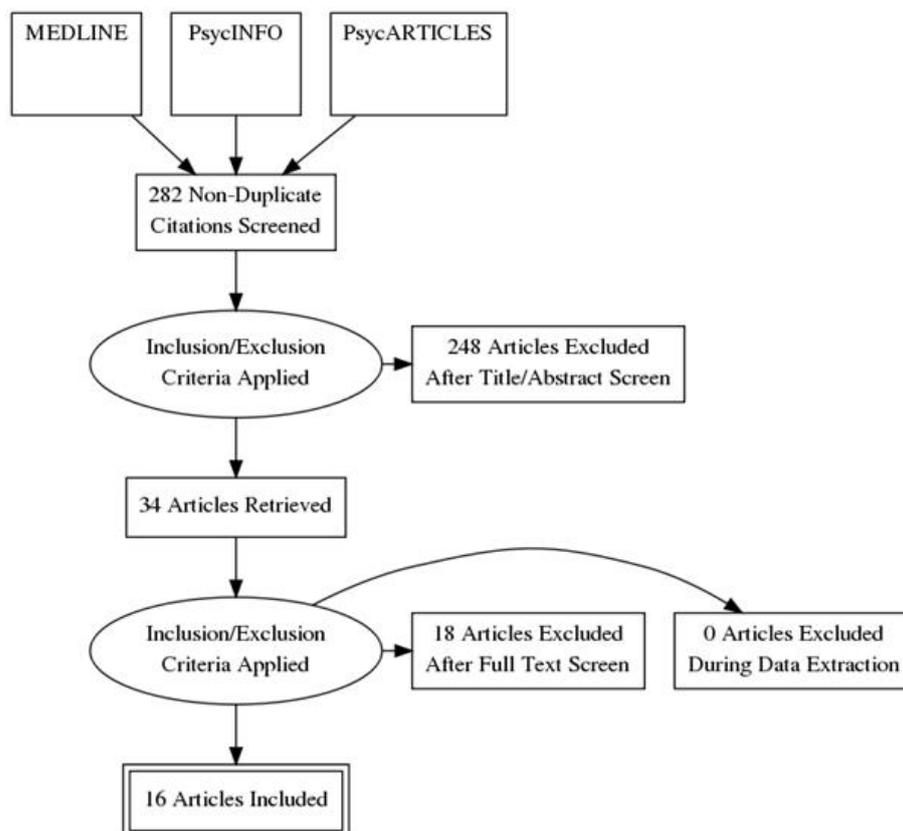
Jack Blake

Introduction

Over 200 000 patients per year are admitted to hospital with head injuries. TBIs are graded as mild to severe based on duration of loss of consciousness, period of post traumatic amnesia, and Glasgow Coma Scale at the scene of the accident. There is growing interest and research into the long-term outcomes following TBI and to what extent a full recovery is made. This literature review sets out to explore the long-term risk of developing the mental health disorders (1) depression, (2) psychosis and (3) cortical dementia following TBI.

Method

EBSCOhost's core medical databases were searched which included: psycINFO, psycARTICLES and MEDLINE for articles relating to TBI and depression or psychosis or dementia. 282 non-duplicate papers were screened finding 16 appropriate for the literature review. Inclusion criteria were that patients must have sustained a TBI and reported either depression, psychosis or dementia at least 1 year post-trauma. Papers looking at predictors of outcome following TBI, relating to recovery of memory, non-head related trauma and a focus on interventions were excluded. On applying the inclusion and exclusion criteria, 34 articles were screened to be appropriate on title and abstract screening. Following this, full-text analysis found 16 articles for inclusion in this review.

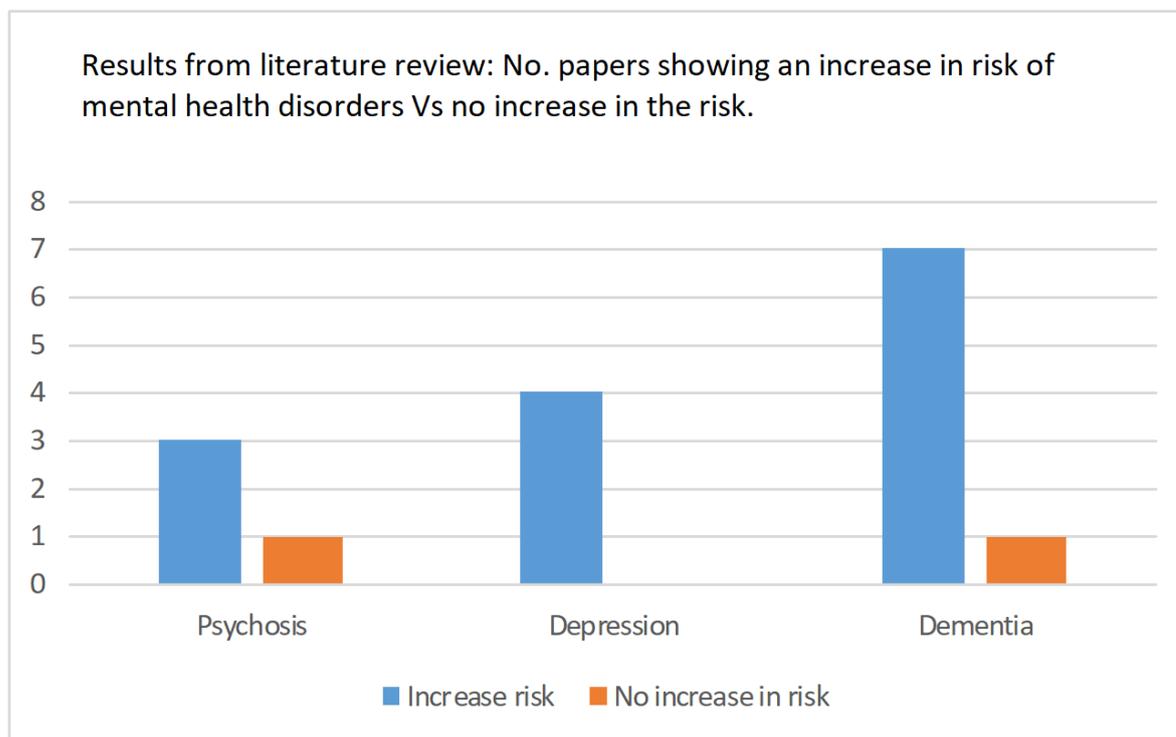


Results

The quality of the studies varied greatly in terms of size – the largest study examined over 20,000 people ¹. The studies were also extremely heterogeneous in terms of the clinical populations studied. The assessment tools used also varied widely and most studies didn't use formalised structural clinical assessment. Four ^{1,2,3,4} out of five studies found a significant increase in the risk of depression following TBI with the other study stating that that post-concussion syndrome can drive depression ⁵.

One of the studies found no increase in risk of psychosis following a TBI ² though three studies found the opposite ^{3,6,7}. Two suggested genetic interplay and the other suggested comorbidities associated with TBI increased the risk. A case series reported on potential link between cannabis use and TBI on psychosis ⁸.

Seven out of eight studies concluded an increased risk of dementia following TBI ⁹⁻¹⁵ with one smaller study disagreeing ¹⁶. Four focused specifically on Alzheimer's disease or pathology whilst the others were more generalised about the dementia.



Conclusion

Despite their differences and their often severe methodological problems, all of the studies suggested that risk of depression is elevated after a brain injury. Whilst many studies suggested risks of psychosis were higher post brain injury, these studies were often small and noted possible interplay of other generic and environmental risk factors. It should also be noted that individuals who are psychotic have higher rates of head injury as a result of their symptoms. Similar issues exist around studies suggesting a possible link between ABI and dementia. Overall, more research is needed to explore this link.

Discussion

This review found good evidence for an increase in the risk of developing depression following a TBI. It is important to note that NICE guidance for follow up of head injury doesn't have any guidance for assessment or managing the psychiatric complications after a brain injury. It seems important to at least warn patients of their increased susceptibility to depression and to educate them of the prodrome.

The evidence base for association of TBI and dementia or psychosis is more nuanced and requires further and better designed longer term studies.

More importantly, this literature review highlights the importance of interdisciplinary cohesion such as that shown in fields like neuropsychiatry. There are patients with clear psychiatric and neurological needs that can fall into placement limbo where there are not established services.

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Jack Blake is a fourth year medical student at Keele University. He has completed his first year of clinical training in Staffordshire including an attachment to a specialist neuropsychiatric service in the Harplands hospital and Bennet centre. Prior to his studies, Jack worked as a social therapist in a medium secure unit from 2014 to 2016 in a therapeutic community setup for the treatment of dangerous and severe personality disorders.

Prevalence of Traumatic Brain Injury among Sexual Offenders: A Systematic Review and Meta-Analysis

Dr Luke Baxter

Introduction

There is growing interest in the association between traumatic brain injury (TBI) and criminal behaviour. Organic personality disorder due to TBI is most often associated with disinhibition and aggression. The development of sexually inappropriate behaviour is a less recognised consequence of TBI.

The prevalence of TBI among sexual offender populations is not well characterized. The objective of this study, therefore, was to determine a meta-analytic estimate of TBI prevalence among sexual offenders, which may inform possible future TBI screening programs for offenders.

Methods

In order to do this, PubMed, PsycInfo and Embase databases were systematically searched to identify papers that measured the prevalence of TBI in a sexual offender sample. A random-effects meta-analysis was conducted to calculate a pooled estimate of TBI prevalence.

Results

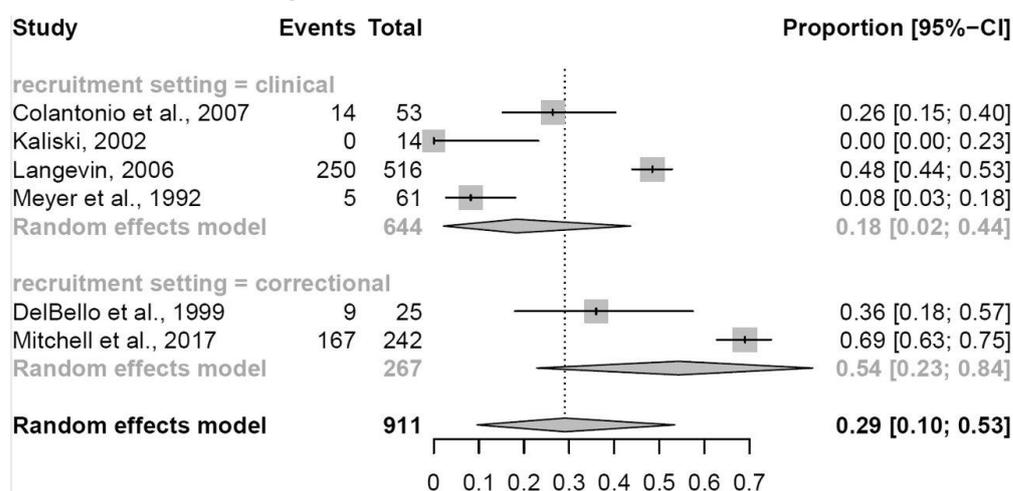
The meta-analytic estimated prevalence of TBI in the sexual offender population, drawn from six studies, was 29.1% (95% CI, 9.6% to 53.5%). This is less than previous meta-analytic estimates for TBI prevalence in the offender population (which range from 51-60.25%), but greater than a previous estimate for the general population (12%). However, when studies were divided according to recruitment setting, those that recruited from a clinical setting had a TBI prevalence of 18% (95% CI, 2% to 44%), whereas those that recruited from a correctional setting had a TBI prevalence of 54% (95% CI, 23% to 84%).

Discussion

To summarise, TBI prevalence appears to be lower among sexual offenders than for the general offender population. However, TBI prevalence among sexual offenders in the clinical and correctional setting are comparable to previous estimates for the general, non-offender and general offender populations, respectively.

Strengths of this study include the systematic nature of the search and the prospective publishing of methods. Limitations of the meta-analysis include the small number of studies that were included and the fact that these studies were generally low quality. There was also evidence of significant heterogeneity between studies. In conclusion, TBI prevalence among individuals in correctional and non-correctional settings appears to be consistent, regardless of the type of crime committed. However, investigations into TBI prevalence for offenders of other crimes should be carried out to validate this finding. These findings support the growing recognition of TBI as a serious health issue among offenders, especially those who are incarcerated. It should prompt greater efforts for the screening of TBI among offenders and the provision of neurorehabilitation for those offenders who are survivors of TBI.

Fig 1. Forest plot showing TBI prevalence among sexual offenders by recruitment setting



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Luke Baxter studied Natural Sciences at the University of Cambridge, specialising in Neuroscience. Following this, he joined the graduate-entry course in Medicine at Cambridge. He graduated in July. He is currently working as an FY1 doctor at the Royal London Hospital in Whitechapel.

Opinion

Why have people with organic mental disorders been systematically excluded from mainstream mental health care?

Hugh Rickards



During my career as a psychiatrist I have witnessed patients with the range of organic mental disorders being systematically excluded from care within adult psychiatry. Examples of this include referrals being refused on the grounds that the disorder is “physical, not mental”, that these disorders were not “core business”, that “we are not trained or confident to diagnose and manage these conditions” and even surprise on seeing these patients at all on the grounds that “I thought you boffins had cured them all”. This talk aims to track this history of this form of social exclusion and to start a conversation about remedy.

Firstly, what sorts of disorders are we referring to? I think the easiest shorthand for this is “anything condition that is referred to in Lishman’s Organic Psychiatry”. This consists of mainly F06 and F07 categories in ICD classification. As an aside, patients with functional neurological disorders have also been excluded from mainstream mental health services, but that is for another time. Why did this exclusion happen? The roots of the problem go back in time to the dualist philosophy of Plato and others, crystallised in western thought by Descartes in the seventeenth century. The very idea that psychiatric disorders could be readily categorized as “organic” or “functional” is problematic. However, throughout the 19th and most of the 20th Centuries, patients with organic mental

disorders were accepted within psychiatric services. Many of us over 50 remember the "chronic organic wards" that were present in all asylums. Patients with the full range of organic mental disorders were cared for on these wards and mental health professionals were exposed to the disorders and developed the relevant clinical expertise as a result.

I think that the modern exclusion of this group of patients had its origin in the social psychiatry movement and was exacerbated by the closure of asylums. During that period, two related ideas were conflated. The first idea, that mental illness was a social construct, was attractive to many including myself and has some validity. This was conflated with the idea that all mental illness was caused by the social milieu (usually oppression at the level of society or the family). This left people with organic mental disorders as an "inconvenient truth". One way in which this contradiction was resolved was to exclude people with organic mental disorders from the category of "mental illness". This has happened in a wholesale manner within many mental health professions, nursing in particular, and the circularity of the argument ("all mental disorders are socially caused, if a mental disorder is caused by an organic factor, it becomes a "physical disorder") persists to this day. Lack of exposure to the conditions then leads to "neuro-phobia" and further increases the chances of exclusion.

There have been two other perpetuating factors. Firstly the provision of community care has become hyper-focused on risk management in the light of several high profile cases of violent behaviour in the community by people with mental illness. This is resource-intensive and has increased the pressure on providers to exclude people with specific diagnoses in order to concentrate on a "core". This change in provision was re-enforced by the National Service Framework for Mental Disorders (2001) which barely mentioned organic mental disorders. Secondly, providers outside of the NHS have taken up the slack and have produced the next generation of "private asylums" around the country in which many of us work. The standards of care in these institutions can be good but the skills are being lost in the NHS along with the ownership of the problem. Finally, the modern neuropsychiatry renaissance may be giving further reason to general psychiatrists to disown the problem. However, relative to the prevalence and complexity of the patients, these services are woefully inadequate.

The question then becomes “what is to be done?”. Does neuropsychiatry want to continue to work towards exclusive ownership of patients with these disorders (which would require a resource revolution and a recognition that more of psychiatric disorders will tend to be regarded as “organic”), or should we be passing the ownership of the patients back to community mental health services and act as their “advisors and supporters”? In general, I would favour the latter plan but the reality will be a hybrid of the two ideas.

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