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healthcare appointments; going to public places; returning to office-based working.

Questionnaires routinely completed at assessment and end of treatment: Montgomery-Åsberg Depression Rating Scale (MADRS); Beck's Anxiety Inventory (BAI); Beck's Depression Inventory (BDI). Adapted treatment with CBT included an extended assessment which helped differentiate anxiety symptoms from ASD. Main CBT adaptations included development of skills for the patient to identify and express emotional experiences and thoughts with the focus on physical sensations and behaviour. Graded exposure items were linked to concrete aims or interests and structured to fit around the patient's routine daily activities. Clinical data was analysed and compared outcomes from the initial standard and subsequent adapted treatment.

Results. The patient's response to the initial course of standard CBT showed a 14% increase in anxiety and 14% increase in symptoms of depression on self-rated measures. The subsequent adapted CBT showed a 31% improvement in anxiety and a 16% improvement in symptoms of depression on self-rated measures. Conclusion. This case report supports literature describing the need to adapt standard assessment and treatment to differentiate experiences related to ASD from discrete anxiety disorders, although there may be some overlap. The promising results support using adapted CBT to ensure appropriate treatment of anxiety disorders in autistic people.

Abstracts were reviewed by the RCPsych Academic Faculty rather than by the standard BJPsych Open peer review process and should not be quoted as peer-reviewed by BJPsych Open in any subsequent publication.

Huntington's Disease and Criminal Behaviour: An Exploration of Psychiatric Risk and Management in a High Secure Forensic Unit

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Aims. Huntington's disease (HD) is an autosomal dominant neurodegenerative disorder characterised by a pathologically prolonged CAG nucleotide sequence in the huntingtin gene (HTT). Neuropsychiatric symptoms such as aggression, depression, impulsivity and psychosis are non-motor signs of HD. The association between HD and criminal behaviour is debated, and evidence lacking. This is particularly relevant in forensic psychiatry, which focusses on the risk assessment of mentally disordered offenders. This manuscript examines the antecedents of offending behaviour in a male diagnosed with HD during admission to a high secure unit, and the evolution of his risk profile from childhood to post-diagnosis. Additionally, through exploration of psychopharmacological management of psychiatric symptoms in HD, this study aims to further our understanding as to how we can best support people with HD in a forensic mental health setting.

Methods. Following review of relevant literature on criminal behaviour in the context of HD, we report the case of a 41-year-old man with a background of dissocial personality traits admitted to a high security unit with symptoms of a delusional disorder; manifesting as paranoia, delusional beliefs and aggression. These were believed to be organically induced within the context of HD, a diagnosis confirmed through genetic testing six months following admission. The patient's symptoms were

only partially responsive to first-line antipsychotics; however, good symptomatic control was achieved with clozapine and sodium valproate, enabling step-down to medium secure specialist services.

Results. In HD patients, there may be a challenge of discerning whether offending behaviour relates to prodromal presentation or whether there are pre-existing antisocial attitudes or behaviour; an uncertainty which was present in this case and within the literature. The age of HD onset is inversely correlated with CAG repeat length, and a longer repeat length has been associated with criminal behaviour. This has the potential for use as a marker to determine the time point in which presenting features are attributable to HD. In this case study it was possible to determine through analysis of the CAG repeat length that the delusional disorder was likely linked to the onset of HD; however, dissocial personality traits were not.

Conclusion. A patient's background relating to the life-course persistence of violence, suicidality and psychiatric symptoms in patients with HD informs the process of formulating their risk profile. Changes to the risk profile also reflect the progressing stages of HD. This highlights the need for awareness of how HD may contribute or predispose to criminal behaviour and how interventions could be targeted during critical periods where they benefit most.

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Ghosts From the Past: A Juvenile Onset Huntington's Disease Case From Bahrain

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Aims. Huntington's disease (HD) is a rare inherited disease in an autosomal dominant pattern, that is most prevalent among Caucasians.

Juvenile onset Huntington disease (JHD) is a rare subtype of the disease, defined by presence of the disease by the age of 20 or younger.

We report a case of a 28-year-old woman with JHD and discuss the challenges we faced in her diagnosis and management. Methods. A now 28-year-old Arab woman, presented to the psychiatric hospital when she was an 18-year-old, complaining of restlessness and low mood. She was diagnosed to be having social phobia and panic attacks, and was given escitalopram. About 6 months after her first presentation, the patient's mother showed up reporting that the patient is doing well without the medication and that she is not going to take them anymore. However, the patient started developing anxiety symptoms two years later and started taking the same medication. Moreover, three years after her first presentation, the patient started developing movement symptoms and mentioned that her father passed away by Huntington's disease. The patient was immediately referred to a genetic lab and a Huntington disease diagnosis was given along with tetrabenazine and risperidone. Moreover, the patient attempted suicide multiple times after worsening of symptoms over the years. A brain magnetic resonance imaging of the patient showed bilateral caudate nuclei atrophy with similar changes affecting the putamen as well but to a lesser extent, changes that are associated with Huntington's disease.

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Results. Juvenile onset Huntington disease is rare, especially among people who are not of European ancestry. Therefore, clinicians are not likely to suspect the illness at an early stage. Late diagnosis not only can prevent patients from receiving the symptomatic treatments that they need, but it can also lead to misdiagnosis. Early referral to genetic testing is required among patients presenting with symptoms and a positive family history. However, risk of suicide is high among patients of Huntington's disease. Conclusion. Juvenile onset Huntington disease is extremely rare. Initial symptoms of the disease could vary and can be misdiagnosed as epilepsy, mood or behavioral disorders, or schizophrenia. Genetic testing is controversial for patients below 18 years old. Having a low suspicion threshold in diagnosing patients with positive family history of HD who are presenting with such symptoms is recommended. There is no cure and treatment is symptomatic.

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Exploring the Potential of Primary ECT Modulation: A Transformative Approach in Schizophrenia Treatment

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Aims. Electroconvulsive therapy (ECT) stands as a crucial intervention for severe and treatment-resistant schizophrenia. Despite being recognized as the most effective acute treatment for severe mood and psychotic disorders, its controversial nature persists due to misconceptions and a lack of familiarity among healthcare professionals regarding modern ECT techniques. This case explores the effectiveness of maintenance ECT in preventing relapse among individuals with schizophrenia, a dimension with scarce existing data.

Methods. A 28-year-old unemployed Caucasian male with treatment-resistant schizophrenia underwent multiple trials of atypical, typical, and depot antipsychotics, yielding no significant improvement in the Positive and Negative Syndrome Scale (PANSS) score. Two attempts with clozapine were hindered by neutropenia. With a baseline PANSS symptom score of 110, the patient struggled with severe auditory and visual hallucinations, preventing coherent conversations. Following 26 sessions of bilateral ECT, the PANSS scale score decreased to 65, prompting transfer to a Transitional Living Facility. After an additional 14 sessions, the patient exhibited significant symptomatic improvement, leading to discharge. The PANSS scale score, after 40 sessions, reached 50. Monthly bilateral ECT sessions and one antipsychotic medication now maintain the patient's reasonably functional lifestyle, encompassing employment, social outings, and assistance in farming with his father. ECT proved highly successful in alleviating both positive and negative symptoms, transforming the patient from severe conversational impairment to independent living and employment.

Results. Empirical data validates clozapine's efficacy for treatment-resistant schizophrenia, yet its clinical use is limited

by the substantial risks of agranulocytosis and neutropenia, relegating it to a third-line option. Neutropenia's onset in our case during clozapine trials prompted a therapeutic shift to electroconvulsive therapy (ECT). Aligned with American Psychiatric Association guidelines, our case underscored ECT's superior efficacy compared with traditional antipsychotics. Acknowledging a 40% non-response rate to clozapine across diverse studies emphasizes ECT's significance as a viable alternative. Despite challenges, contemporary ECT methods promise to overcome traditional constraints, reduce stigma, and improve treatment accessibility.

Conclusion. This case underscores the potential benefits of ECT as a valuable treatment modality for individuals with treatment-resistant schizophrenia, effectively managing both positive and negative symptoms and significantly improving daily functioning. The success observed in this case suggests that monthly bilateral ECT and one antipsychotic medication can play a crucial role in enhancing the quality of life for patients with treatment-resistant schizophrenia.

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From Irritability to Amnesia: Unraveling Thalamic Glioma – a Case Report

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Aims. Gliomas, encompassing astrocytomas, oligodendrogliomas, and ependymomas, constitute the majority (40–55%) of primary brain tumors. Diagnosis can be challenging due to their uncommon nature, subtle symptoms, and diverse clinical manifestations. While neurological signs are typical, psychiatric symptoms may occasionally precede them. This case report explores a 59-year-old man whose initial psychiatric symptoms, resistant to treatment, evolved into memory impairment, ultimately revealing a highgrade glioma in the thalamus.

Methods. A 59-year-old male patient presented to the psychiatric service, expressing concerns about excessive anger and aggression. His family observed his behavior as abnormal, noting uncharacteristic personality changes, particularly increased irritability. Following an outpatient psychiatric evaluation, he was diagnosed with excessive irritability. Over time, the patient's aggressive behaviors intensified, accompanied by feelings of being ignored and devalued by his family, heightened emotional sensitivity, and episodes of muteness. Despite two trials of medication (i.e., sertraline and alprazolam), there was a deterioration in adaptive functioning. Two years after the first onset, the patient experienced unfamiliarity with surroundings, forgetting place names, memories, and people's names. The patient had no family history of neurological or psychiatric illness, and there was no evidence of substance use in his past. To rule out organic causes, an MRI revealed a 17×21 mm lesion in the right thalamus and a calcified focus in the superior part of the left tentorium. Subsequent biopsy confirmed a high-grade glial tumor: anaplastic astrocytoma Grade III, with a Ki-67 index of 10%.

Results. The extended onset of memory impairment in our patient, following a 3-year history of aggressive attacks and irritation, prompts an exploration of the intricate interplay between