a private NHS funded in-patient adolescent psychiatric unit, where she was placed on Section 2 of Mental Health Act. After two months she was transferred to an out of area psychiatric bed. While on home leave she took another overdose and needed admission in High Dependency Unit. When medically fit she was moved back to psychiatric unit. After two months she was moved to a local in-patient unit. Due to dietary restriction she was commenced on nasogastric feed. At that point the local psychiatric unit could not provide the support she needed and she was transferred to another in-patient adolescent unit in the region. After being an in-patient for 18 months when her condition improved a discharge planning meeting took place. She was still on nasogastric feed and sometimes had to be restrained. The local and regional in-patient and crisis consultants suggested that she should be discharged under Community Treatment Order. The community consultant took legal advice. The legal advice was that the provision of nasogastric feeding in the community without young person's consent would be likely to be unlawful and a violation of her Article 3 rights under the European Convention of Human Rights. Such restraint would be likely to amount to an unlawful deprivation of liberty and a breach of her Article 5 rights under the European Convention of Human Rights.

Results. It is deeply concerning that she had to be moved between four in-patient units during one episode of in-patient admission due to lack of appropriate bed availability. Due to her age and complexities in the case, legal advice had to be taken because the consultants involved failed to agree on the appropriate application of legal framework.

Conclusion. This case clearly highlights the need to address the issue of adolescent in-patient psychiatric bed shortages as well as the importance of educational programmes aimed at improving the knowledge and skills of professionals on the application of legal framework in children and adolescents.

Full Remission of Obsessive Compulsive Disorder (OCD) Symptoms in Huntington's Disease (HD) Using Fluoxetine

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Aims. HD is an autosomal, dominantly inherited, neurodegenerative disorder which can present with cognitive, motor and behavioural symptoms. Recent studies suggest that obsessive compulsive disorder (OCD) symptoms, although not common, may precede or coincide with symptoms in patients with HD. We present a case of an adolescent boy presenting with symptoms of OCD, for 4 months duration, in background of three years diagnosis of HD.

Methods. A 15-year-old boy from South India, presented with recurrent, intrusive thoughts of sexual content, consistent with obsessions and some instances of compulsions in the form of avoiding to do deviant sexual act like fetishism, and having excessive worries about an act he had done earlier for 3 months duration (supported by high scores on Yale-Brown Obsessive Compulsive Scale; Y-BOCS). Patient had normal birth and development and had no past history of psychiatry disorder, however there was family history of HD in multiple first and second-degree relatives. He was on treatment for movement symptoms of HD, diagnosed 3 years back and was on Tetrabenazine for 2 years. Initial psychiatric assessment found the symptoms to be consistent with OCD due to Huntington's disease, according to Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5). The patient was admitted to the mental health unit and was started on Fluoxetine, titrated to a dose of 20mg daily for symptoms of OCD.

Results. Subject showed an excellent response to fluoxetine with complete remission of OCD symptoms within 4 weeks of treatment. The relationship between OCD and HD has been little-investigated, despite the fact that both diseases are associated with striatal dysfunction and that the number of case reports of obsessive-compulsive symptoms either preceding the clinical onset of HD or during later stages of the disease is increasing. For example, Dewhurst et al. reported "obsessional features" in 7 of 102 patients at onset of HD.

Conclusion. Firm conclusions to explain this result cannot be drawn. However, a hypothetical involvement of the serotonergic system, suggested by the excess of OCD, seems supported by the response of said subject to fluoxetine. It may be worth further exploring the value of the psychiatric picture in selecting the appropriate treatment for at least some cases of HD. Anecdotal evidence suggest that SSRIs alone or in combination with atypical antipsychotics like olanzapine may be useful for these patients. However, these hypotheses need further testing.

A Case of Urticaria One Month After Initiating Fluoxetine

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Aims. According to NICE guidelines, fluoxetine is a first-line antidepressant for the management of depression in children and adolescents. There has been little discussion on urticaria occurring after 4 weeks on fluoxetine. Urticaria is defined as an itchy skin lesion "with localised oedema and is surrounded by redness which is also known as a wheal and flare phenomenon". In most urticarial scenarios, the trigger which is usually an allergen leads to an increase in levels of histamine among other chemicals into the skin.

Methods. We present the case of a 16-year-old young person with no previous history of skin conditions, who developed urticaria 4 weeks after starting fluoxetine for depressive disorder. No other trigger could be identified. On discontinuing fluoxetine, the rash gradually declined over time and completely resolved ten days later. On further enquiry, the patient reported eating at least one chocolate bar a day. He did admit to receiving a large amount of chocolate just before the rash began and hence was possibly eating more than his usual amount at the time. Furthermore, the rash occurred at the point when steady-state levels of fluoxetine were believed to have just been reached.

Results. Adverse effects of medication have always been a challenging part of managing patients. Although rash that develops acutely after starting fluoxetine has been published so far, literature on rash that develops after 4 weeks on treatment is limited. Cederberg et al (2004) and Gahir (2021) described the association

of SSRIs (fluoxetine and sertraline) and chocolate (which contains serotonin) leading to an itchy hive-like rash. In these cases, discontinuation of the SSRI and use of antihistamine led to a resolution of symptoms.

We report a case who developed urticaria 30 days on fluoxetine without any other identifiable triggers. Aspects of this case to support a possible rash caused by chocolate-fluoxetine interaction include the rash occurring when the patient was consuming chocolate (quantities possibly increased immediately prior to the onset of rash), rash occurring when steady-state levels of fluoxetine had just been reached, no other identifiable trigger to explain the rash in the history and the slow resolution of the rash which can be explained by the long half-life of fluoxetine.

Conclusion. This report highlights the importance of being mindful of this rare dermatological side effect of fluoxetine despite it occurring weeks after initiation. Patients should also be made aware of this possible side effect and its association with consuming chocolate.

Rapid Cycling Bipolar Affective Disorder After COVID-19 Infection Accompanied With Neurological Symptoms

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Aims. This case highlights an atypical presentation of a patient with known history of Bipolar Affective Disorder who experienced rapid mood changes and atypical neurological symptoms after he was tested positive for COVID-19.

Methods. Here we present a 63 years old male patient who was an inpatient in low secure forensic unit and has a history of Bipolar Affective Disorder. Patient reported that he started to experience COVID-19 symptoms and was tested positive on 12th April 2020. It was observed that patient experienced low mood, flat effect, anhedonia and decreased appetite for more than a month after he was tested positive. According to his medical records, he experienced significant mood changes suggesting major depression and manic/hypomanic episodes, 4 times to be specific, over 6 months period after having diagnosed with COVID-19 which is correlated with diagnostic guidelines for Rapid cycling Bipolar Disorder. Patient was observed to experience 1 major depressive episode over period of 6 months before his COVID-19 diagnosis. He also reported experiencing neurological symptoms such as tremor, numbness and unsteadiness on one leg. Although it was found that his lithium level was above therapeutic range at the beginning of these symptoms, even after successful reduction of Lithium dose, patient continued to experience these symptoms for another month. There were no gross abnormalities in physical examination and his blood results were not significant. In addition to Electroencephalogram (EEG); Computed Tomography (CT) scan, Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA) were conducted and the results were all insignificant. During this time, he was fairly compliant with his medications. Additionally, his mood was stabilised only partially with the medications he was taking. He did not have any other major environmental, psychological or physical changes that might explain his rapid mood cycling.

Results. Authors considered various different causes for this patient's fluctuating mood. One confounding factor that was considered was blood lithium levels. However, that was proven to be irrelevant since patient continued to experience mood changes and neurological symptoms with therapeutic lithium levels. Also no other organic reasons were found that could explain his neurological symptoms.

Conclusion. Although, authors consider that longer observation period and other confounding factors could affect findings, they cannot confidently reject the impact of COVID-19 infection on patients with enduring mental illness and recommend further research which could lead to more comprehensive guidelines

The Use of Genetic Testing in the Management of Depression

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Aims. We report on a case of depression where genetic testing was used to determine potential treatment modalities.

Methods. The patient is a 78-year-old man who had suffered from depression for 55 years. He had a serious episode in 2002. He developed a further depressive episode in 2018 which did not respond to paroxetine. He was offered TMS and was initially treated in the NHS and subsequently in the private sector. He went into remission with TMS and continues to remit with TMS however his depression became unstable and it was clear that the paroxetine was having no effect. He agreed to have a genetic test, a buccal mouth swab was taken and posted to gensense in the United States. An 18 page document and a half hour session with gensense are included in the cost of the test. The results of his genetic test and suggestions regarding treatment are detailed below.

SLC6A4 L(G)/S serotonin transporter indicating a less favourable response to SSRI medication (20% response versus 40% response). SNRI medication may be useful.

BDNF Val/Met Met carriers may have poor response to SSRIs and an improved response to SNRI's and TCA's. Met carriers have a 3 times better response to exercise than Val/Val

MTHFR A/A variant, this results in a 70% reduction) in the ability to convert folate to methyl folate (required for the manufacture of serotonin). Taking L-methylfolate supplementation (7.5mg) may improve serotonin production and provide a 2 times increase in response rate to antidepressants.

COMT Val/Val variant indicates improved response with brain stimulation therapy such as ECT and TMS

CACNA1C A/A variant which increases the anteromedial and amygdala activity and increased neuronal activity as a result of increased calcium channel receptors. This variant is associated with more depression, OCD and anxiety. Using lithium, sodium valproate and lamotrigine could be potentially useful in this group.

Results. The patient's antidepressant was switched from Paroxetine to Venlafaxine XL 150 mg, he started taking L methyl folate supplements (7.5mg daily) and was put onto sodium valproate 250 mg 3 times a day. His HAM-D went from 39 in

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