depression. Shown to improve emotional processing and engagement in therapy.

4. Enhancement of Social Skills.

Studies indicate that art therapy significantly improves social interactions, particularly in patients with learning disabilities.

Conclusion: Art therapy is a valuable adjunct that provides alternative communication channel, especially for non-verbal or emotionally withdrawn individuals.

Clinical Implications and Future Directions:

Incorporate art therapy into multidisciplinary-treatment plans for patients with learning disabilities.

Investigating neurobiological mechanisms underpinning art therapy's impact could optimise therapeutic approaches.

When Mind and Body Speaks: Understanding Dissociative Neurological Symptom Disorder-ICD–11

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Aims: Dissociative Neurological Symptom Disorder (DNSD), also known as Conversion Disorder, is a common diagnosis among mental health patients in Pakistan. Despite its prevalence, research on DNSD, especially regarding patient experiences, is limited. Family-related stressors are significant contributing factors in its development, with familial discord playing a key role in triggering symptoms.

Methods: A 24-year-old female was referred by a neurologist after presenting to the outpatient department in a wheelchair due to a fear of falling. She reported symptoms including jerky body movements, episodes of apparent loss of consciousness, diarrhoea, weakness, visual disturbances, headaches, and palpitations, which had persisted for over two years. Despite multiple consultations, no organic cause was identified. She had been prescribed various medications with no improvement. Upon evaluation, a diagnosis of DNSD was made, compounded by significant emotional stress, particularly familial discord. A multidisciplinary approach, involving specialists in neurology, ophthalmology, ENT, and gastroenterology, helped rule out underlying physical conditions. Her pre-morbid history indicated high academic achievement but chronic familial stress.

Results: This case emphasizes the importance of recognizing psychosomatic presentations in patients with unexplained neurological and physical symptoms. Despite extensive negative investigations, the patient's psychological stressors, such as familial discord and fear of stigma, were key contributors to the onset of her symptoms. The patient's fear of walking further exacerbated her physical disability. Treatment included sertraline, mirtazapine, and short-term benzodiazepines for anxiety and depression. Psychological therapies such as Cognitive Behavioural Therapy (CBT), deep breathing, safe-place visualization, imaginal exposure, systematic desensitization, and gratitude journalling were used to address emotional issues and reduce disability. This comprehensive approach resulted in symptom resolution, and the patient was symptom-free within 18 months, now pursuing an MPhil degree.

Conclusion: This case highlights the value of a multidisciplinary approach in diagnosing and treating complex physical symptoms with unclear aetiology. Addressing underlying psychological stressors and utilizing appropriate psychosocial interventions significantly improved the patient's condition. Early identification and holistic management combining medical, psychological, and emotional support are crucial for effective treatment of DNSD.

Sodium Amytal – No Longer Prescribed, but Still Relevant (And Dangerous!)

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Aims: Sodium amytal is a barbiturate medication, first synthesised in Germany in the 1920s to treat anxiety and sleep disorders; as well as being used as an anaesthetic. This case report discusses sodium amytal prescription and subsequent dependence in an 82-year-old female with a history of anxiety and agoraphobia. It aims to highlight historical indications, mechanism of action of and potential dangers of cessation. As such, clinical management of withdrawal is discussed including the initial use of a more commonly prescribed barbiturate in place of amobarbital; with the ultimate aim being to cease such dangerous medication and to consider safer alternatives – pharmacological and psychological.

Methods: Mrs F was referred to Older Persons Psychiatric Services in 2024. At this time, her GP reported that she was 'one of only 30 patients in the UK' to be prescribed sodium amytal, and that it was 'no longer being produced'. At the time of referral, Mrs F had just a 7-day supply of medication left.

Guidance from the local Medicines Information team had been sought prior to admission. They recommended a phenobarbital taper for seizure prophylaxis when discontinuing other barbiturates, where 100 mg amobarbital was roughly equivalent to 30 mg phenobarbital. Phenobarbital is a long-acting barbiturate which would be more commonly found in practice, given its use in management of epilepsy.

As such, an attempt was made at the safe withdrawal of sodium amytal in the inpatient setting. This was due to the significant risks associated with abrupt withdrawal, including seizure, hallucinations and cardiovascular collapse tending to occur after 16 hours of cessation with such risk remaining up until approximately 5 days.

Mrs F had run out of medication on the day that she was admitted, and given the aforementioned risks, a thorough plan was developed to ensure close monitoring. She was commenced on 1:1 special observations initially due to the risk of seizures. Her clinical observations were performed on a regular basis and a CIWA (Clinical Institute Withdrawal Assessment for Alcohol) score was also performed hourly to monitor withdrawal severity. To this end, lorazepam was prescribed at a dose of 1 mg every 6 hours, as well as an 'as required' dose of 1 mg prescribed for when CIWA score was >8.

At consultant review the following day, Mrs F was commenced on phenobarbital at a dose of 30 mg QDS + 30 mg PRN to a maximum of 150 mg/day, continuing on regular lorazepam at 1 mg QDS. Of note, this is slightly lower than the aforementioned recommendation which would equate to 162 mg/day. Subsequently, owing to increasing agitation and anxiety, Mrs F's dose was increased to 60 mg mane, 30 mg TDS plus 30 mg PRN and her dose of lorazepam

was reduced to 1 mg TDS. She was discharged on both medications, 9 days after admission.

Results: The treating team discussed the preferred option to follow on from the withdrawal of amobarbital with subsequent tapering of phenobarbital, given that it shares many of the same risks as the former. Mrs F however was not keen to change medications again at present, owing to her age and ongoing anxiety. In fact, at a subsequent outpatient review, Mrs F reported that she felt that she would require more lorazepam to cope with ongoing anxiety, however this was not facilitated. In terms of patient safety, it should be highlighted that the patient was discharged on 2 addictive, sedative medications (even if the lorazepam was a short-term measure), which could prove challenging to rationalise moving forward and perhaps gives an insight into the ongoing difficulties faced by outpatient teams in terms of cessation of addictive medicines. Importantly, the patient would not however experience the same supply issues currently with phenobarbital. It should also be noted that she had declined psychological therapy with the Community Mental Health Team for Older Persons.

Conclusion: The aims of this report were to highlight the use and action of the barbiturate class of medications, whilst also discussing difficulties with supply issues, dependence and the associated potential for withdrawal. Ensuring patient safety is paramount, and in many cases patients who are prescribed this class of medications for a prolonged period of time require inpatient management to allow for a safe transition to an alternative medication.

Ketamine Cystitis Following Ketamine Therapy for Treatment Resistant Depression – Case Report

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Aims: Ketamine is a novel and exciting putative antidepressant medication for patients with treatment-resistant depression. A complication commonly seen in frequent and heavy recreational use of ketamine is ulcerative cystitis, which presents with lower urinary tract symptoms (LUTS) and upper renal tract damage and can be seen in over 25% of regular users.

Although ketamine-induced cystitis (KIC) is a recognised complication in recreational use of ketamine, its occurrence in therapeutic use of ketamine in depression has so far not been reported. The exact pathogenesis of KIC is currently unknown, making treatment and prevention advice much more difficult. Early diagnosis of KIC and immediate cessation of ketamine has been shown to improve adverse urinary tract symptoms and prevent further damage.

Methods: We present a case of a 28-year-old female who was started on ketamine treatment for depression, and who then developed symptoms of KIC, which was confirmed by urine microscopy, culture and analysis.

Results: Ketamine-induced cystitis (KIC) typically starts with urinary symptoms that include dysuria, urgency, nocturia and urinary frequency. With continued use, symptoms can progress to incontinence, haematuria, bladder wall fibrosis and ulcerative cystitis. Ongoing use of the drug can lead to involvement of the upper renal tract, including hydronephrosis and chronic kidney failure. Physical examination and investigations may demonstration suprapubic pain, sterile pyuria and increased eosinophils within the bladder wall. The pathophysiological mechanisms of LUTS are not yet fully understood and further research to define therapeutic options would be useful.

Imaging of the bladder in severe cases may demonstrate a grossly constricted bladder with thickened walls. Cystoscopy often demonstrates a friable bladder mucosa that is prone to bleeding. Microscopically, the urothelium may appear denuded, ulcerated and infiltrated by inflammatory cells, such as mast cells and eosinophils. Other findings include submucosal fibrosis, muscle hypertrophy and collagen deposition.

Although the exact pathogenesis of KIC is not yet fully understood, various mechanisms have been postulated and it is likely that several pathways are involved simultaneously.

One theory is that the ketamine and its metabolites (which are largely excreted by the urinary tract), cause direct toxicity to the bladder. These disrupt the urothelial integrity of the bladder epithelium and initiate interstitial fibrosis. This has been demonstrated in animal models and the level of damage directly correlates with the dose of ketamine used.

Another theory is an IgE-mediated response. Bladder samples in ketamine users frequently show raised inflammatory cells and messengers, including mast cells, eosinophils, COX-2 (cyclooxygynase-2), NOS (nitric oxide synthase) and IgE. These levels fall once the patient is in remission from ketamine use and rise again once ketamine is restarted. This suggests an inflammatory response or a hypersensitivity reaction leading to bladder damage.

Ketamine can also directly stimulate various chemicals, including adenosine triphosphate, antiproliferative factor and oxidative stressors, which subsequently lead to changes in the bladder wall. It has been reported that the NMDA receptor (NMDAR) and angiogenic factors can also cause microvascular injury within the bladder.

Other proposed theories include aberrant neurotrophic factors, protein kinase B, mTOR pathways, metadherin and MAPK pathways, leading to downstream fibrosis of the bladder.

Early diagnosis of KIC and immediate cessation of ketamine use has been shown to improve symptoms, reverse early disease and prevent further damage.

Conclusion: To our knowledge, this is the first reported case of KIC in a patient receiving treatment-dose ketamine as part of their antidepressant therapy.

A Clinical Conundrum in Anorexia Nervosa – Starvation Hepatitis vs Refeeding Hepatitis

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Aims: Anorexia nervosa (AN) is a severe eating disorder, with a lifetime prevalence estimated to be 0.3–0.9%. Transaminase elevations are common in patients during hospital admission, reaching a prevalence of 43%. Although usually caused by refeeding, prolonged starvation can also cause an exacerbation of liver enzyme levels. It is important to differentiate between the two, as the treatment plans are quite different. We herein present a report of a 34-year-old lady with anorexia nervosa, who presented with extreme