The treating team re-commenced oral clozapine to which she remained initially non-compliant due to catatonic features. With advice from the specialist psychosis services a few doses of intramuscular clozapine was used to facilitate re-titration. Following regular compliance and optimisation of oral clozapine, there was significant remission of clinical symptoms, with patient returning to her baseline mental state and functioning. During the period of admission, platelet counts were closely monitored which kept fluctuating reaching sometimes below 30×109/L without any clear association with clozapine dose. No bleeding symptoms or signs were ever reported.

Results. Clozapine is a medication with haematological side effects; however, low platelet count is very rare. This patient ultimately underwent bone marrow biopsy which established Immune thrombocytopenia. She was discharged to the community with a plan of continuing clozapine, close monitoring of blood count and regular follow-up with haematology services for further clinical management.

Conclusion. Careful clinical evaluation and timely investigation is important to establish the cause for side effects before associating it with clozapine and discontinuing the treatment. This helps in ensuring continuity of clozapine in patients who clearly benefit from long-term use of clozapine.

'Moon Shot': A Case Study of Augmentation of Clozapine With Fluvoxamine in an Adolescent With Treatment-Resistant Schizophrenia

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Aims. Very early onset schizophrenia is considered a rare and severe form of schizophrenia, with onset before age 13. Early initiation of antipsychotics significantly improves outcomes and prognosis. Treatment refractory status is not uncommon and clozapine currently remains the most effective option in this scenario. However, approximately 40–70% of antipsychotic-resistant patients do not respond, or respond only partially to clozapine. Additionally, many patients stop clozapine due to side effects, many of which are due to its active metabolite, nor-clozapine. Since clozapine-resistant patients have limited alternative treatment options, augmentation strategies must be considered.

This case study highlights one such augmentation strategy using fluvoxamine. Fluvoxamine inhibits CYP450 1A2 isoenzymes reducing the risk of the metabolite induced side effects and synchronously increasing plasma concentrations of clozapine.

Methods. The case study is of a 13-year-old female diagnosed with paranoid schizophrenia characterized by second and third person auditory hallucinations, delusions of persecution, paranoid pseudo-community, impulsive aggression and cognitive decline. She screened negative for developmental disorders, metabolic and genetic anomalies and medical co-morbidities. She had failed trials of two atypical antipsychotics. Clozapine was subsequently initiated and optimized to 500 mg/day (Serum Clozapine of 981 mcg/L). Partial improvement in symptomatology was observed. However, dose adjustments were difficult throughout due to side effects of clozapine and pharmacological agents such as Metformin, Lamotrigine, Ipratropium Bromide and Propranolol were used prophylactically to mitigate the side-effects. The polypharmacy, social isolation, excessive sedation and emerging obsessive-compulsive symptoms contributed to secondary negative symptoms. Low-dose fluvoxamine was subsequently used as an augmentation strategy following which improvement was noted.

Results. Several studies have shown that co-administration of fluvoxamine may increase the steady-state serum concentrations of clozapine by a factor of 5. Optimizing the serum ratio of Nor-clozapine and clozapine levels, thereby, reduces the need for aggressive polypharmacy. Low doses of fluvoxamine inhibit the CYT activity, enough to raise the level of clozapine even when the dose of clozapine is reduced by 50% which is the target going forward for this patient.

Conclusion. Although current practice guidelines recommend clozapine mono-therapy for treatment resistant schizophrenia, augmentation of clozapine with fluvoxamine can be considered for those who do not respond adequately to clozapine mono-therapy or cease treatment due to its side effects. However, considering the unpredictable effect on clozapine plasma levels, concomitant use should ideally be initiated in facilities like a pediatric intensive unit where close surveillance is possible especially for side effects such as myocarditis especially in adolescents.

Effect of Escitalopram on Glycemic Control and C-Reactive Protein in Patients With Depression and Co Morbid Type 2 Diabetes Mellitus – a Study on Indian Population

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Aims. There is a bidirectional link between Depression and type 2 Diabetes mellitus (T2DM). Treatment of depression with selective serotonin reuptake inhibitors (SSRIs) may improve glycaemic control and may be beneficial for patients with comorbid depression and diabetes mellitus. The aim of the present study was to assess the effect of escitalopram on C-reactive protein (CRP) and glycaemic control in patients with comorbid T2DM and depression.

Methods. A prospective interventional follow up study was conducted in a tertiary health care institute in urban India. Adult males and females who were diagnosed with Type 2 DM, having depression as per ICD-10 and treatment naïve for both the disorders were included for the study. Participants with other psychiatric disorders, on thyroid medication or on any medication that can have effect on CRP levels, having history of any infection/allergic or inflammatory conditions were excluded from the study. Sociodemographic details were collected. The severity of depression was assessed using Hamilton Depression Rating Scale (HDRS) at baseline. Escitalopram was started and titrated upto required doses for each patient. Levels of fasting blood glucose, post prandial blood glucose, HbA1c (Glycated Hemoglobin) and CRP were also measured at baseline. At the end of 3 months, severity of depression scores and blood levels of above mentioned

parameters were measured and compared with their baseline values.
Results. A total of 125 patients (females-n = 70, males-n = 55) were included for the study. The mean age of the sample was 63.2 years (SD 10.6). Most of the participants were educated and employed. The mean HRDS score of the participants at baseline and at three months was 20.3 (SD 3.7) and 18.0 (SD 3.9) respectively. The mean HbA1C of the participants at baseline and at three months was 8.4 (SD 1.2) and 7.8 (SD 1.2) respectively. The mean CRP of the participants at baseline and at three months was 4.0 (SD 5.6) and 2.8 (SD 4.3) respectively. There was significant reduction in depressive symptoms (Z score= -6.894, P value <0.05), levels of HbA1C (Z score= -7.936, P value <0.05) and CRP levels (Z score= -6.158, P value <0.05) at follow up after treatment with escitalopram. No significant correlation was observed in these parameters across gender.
Conclusion. Treatment with escitalopram reduces the severity of depression and the ongoing inflammatory process amongst these patients.

SILENT Syndrome – a Case of Lithium Neurotoxicity on Maintenance Therapy

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Aims. Lithium is licensed to treat bipolar disorder, which is characterized by recurrent episodes of depression and mania/hypomania. It is also used as an adjunctive medication in patients who have inadequately responded to first and second line treatments of unipolar depression. Lithium has a narrow therapeutic index and the potential for toxicity requires levels to be closely monitored, particularly during any intercurrent illness or initiation of new medications. There is a rare but important effect of lithium toxicity of which there is little awareness: the Syndrome of Irreversible Lithium Effectuated Neurotoxicity.
Methods. A 57-year-old lady presented to the emergency department with a ten-day history of vomiting, diarrhoea, and abdominal pain. She had a history of recurrent depressive disorder managed with fluoxetine and lithium for ten years. On presentation, she was hypovolemic and required resuscitation with I.V. fluids. Clinical examination revealed significant ataxia and myoclonus. Neurological examination was limited by her inability to follow commands. She was orientated to person but not time or place. A collateral history was obtained from her husband. He reported a 3-day history of increasing confusion on a background of a 6-week history of gradual functional decline. History and examination were concerning for lithium neurotoxicity. Lithium level = 2.6mmol/L (0.4–0.8mmol/L), indicating lithium toxicity. Deterioration in renal function from baseline - urea 14.2, creatine level 120 umol/L eGFR 43mil/min. There was no evidence of infection, full blood count and CRP were within normal parameters. MRI brain showed mild degree global volume loss consistent with chronic small vessel microvascular ischaemia. She was commenced on haemodialysis in order to rapidly reduce her serum lithium levels.
Results. Lithium levels post haemodialysis were 1.2mmol/L and within days fell to <0.4mmol/L. Further lithium treatment was held during admission, but she continued to exhibit signs of neurotoxicity. Two weeks post-admission her confusion persisted (MOCA 13/30). She remained tremulous and ataxic. A diagnosis of Syndrome of Irreversible lithium-effectuated neurotoxicity (SILENT) was made. She required intensive physiotherapy and occupational therapy input. 8 weeks post admission she had returned to her cognitive baseline and was mobilising independently.
Conclusion. SILENT syndrome is a rare consequence of lithium toxicity secondary to elevated lithium levels in the central nervous system which if sustained it is thought can lead to cerebellar demyelination as was evidenced in this case by our patient's symptoms. The insidious onset of her lithium toxicity in the community led to a prolonged period of toxicity that went undetected. No clear precipitating factor was identified. Vigilance is required for toxicity and this case highlights the importance of family members being aware of the signs. A patient when confused may no longer be able to advocate clearly for themselves or seek appropriate medical attention. The patient also delayed consulting with her GP as her GP practice was located an hour and a half from her home and she could not secure an appointment during the summer months. Patients prescribed lithium require timely access to GPs for monitoring and consultations. Despite her experience with toxicity, the patient opted to restart lithium due to a recurrence of depressive symptoms. She is adhering to close monitoring of serum lithium levels. The patient and her family received thorough psycho-education regarding symptoms and signs of lithium toxicity.

Fahr's Disease (Primary Basal Ganglia Calcification) and Violence: Case Report and Literature Review

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Aims.

Background:
Fahr's disease is a rare and complex neuropsychiatric disorder resulting from abnormal calcium deposition in the basal ganglia and cerebral cortex. It can have a profound impact on an individual's social functioning as well as causing a wide variety of neurological symptoms, cognitive deficits and motor impairment. A number of specific mutations have recently been identified in phosphate transporter and other genes, but around half of all cases have unidentified mutations. Impulsivity, aggression and violence may pre-date the other manifestations of the illness.
Methods.

Case Report:
Patient X is a 58 year old man currently detained in an independent hospital locked rehabilitation unit following the breakdown of a care home placement. His first admission to hospital was at the age of 18 when he was diagnosed with mania. He had multiple further hospital admissions as well criminal convictions for acquisitive and violent offences. In 2005 he threatened to stab a stranger if he did not give him a cigarette and he was arrested and admitted to a medium secure unit under Section 37 with diagnoses of bipolar affective disorder and emotionally unstable