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