EPV1605

Desvenlafaxine-a causative agent of extrapyramidal side effects

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Introduction: Extrapyramidal side effects due to antipsychotics is very common but antidepressants being the causative factor is very less studied. Among antidepressants escitalopram is the most commonly reported. SSRI's most commonly cause extrapyramidal side effects than other antidepressants. The major theories are changes in chemical, anatomical and physiological perspectives of neurological system. Reported cases shed light that akathisia occurs most commonly followed by dystonia, parkinsonism and tardive dyskinesia states in antidepressant induced extrapyramidal side effects. Desvenlafaxine (o desmethyl venlafaxine) inhibits reuptake of dopamine, serotonin and norepinephrine. EPS occurs due to inhibitory effect of serotonin on dopaminergic pathway in striatum. Females suffer from it more commonly than men. Increasing age in women,CYP2D6 inhibition by concomittently used drugs can increase the risk. SNRI's less frequently cause EPS than SSRI's. Although desvenlafaxine is very well tolerated this rare side effect increases noncompliance and chances of suicide. Drug induced parkinsonism also predicts future chances of parkinsonism. Usage of desvenlafaxine sometimes present to the emergency department as dystonia causing panic among care givers of the patients.

Objectives: To determine desvenlafaxine's role in causing extra-Pyramidal side effects

Methods: We report here 8 cases of desvenlafaxine induced extrapyramidal side effects. All follow up cases of depression coming for follow up to dept of psychiatry IMS & Hospital who were on desvenlafaxine was analysed.the patients developong extrapyramidal side effects were detected and detailed evaluation and appropriate management was done for those specific cases..all these cases were collected over a period of last 4 years.

Results: In our case series we bring into light rare occurences of extrapyramidal side effects due desvenlafaxine. 5 out of 8 cases were females.most of the symptoms of developed within 5 days of starting the medicine.4 of these cases resulted in secondary parkinsonism, 3 of them resulted in akathisia and one resulted in acute dystonia post adminstration of desvelafaxine. The average dose of desvenlafaxine in all the cases were within 50-100mg. When after extrapyramidal side effects desvenlafaxine was withdrawn replacement with mirtzapine, escitalopram, sertraline or duloxetine was used instead of it resulting in good symptom reduction of primary illness.

Conclusions: Extrapyramidal symptoms with desvenlafaxine is extremely rare. In our case series we highlighted the importance of a keen eye to check for Extrapyramidal side-effects even with the administration of Antidepressants. Future reasearch is needed to find predictors and exact mechanism of action for the same.

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Lymphopenia without neutropenia in Clozapine treatment. A review and a case report

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Introduction: In 1975 Clozapine was retired after 16 cases of severe neutropenia, with a mortality of 50%. It wasn´t until 5 years later when its effectiveness in treatment resistant schizorenia, with a mandatory hematological follow up.

In studies available we find that clozapine treatment is related to neutropenia and not leukopenia. In the case we present below neutrophils are within range, but it´s lymphocytes that are affected.

Objectives: We hope that our expirience, and review can help other professionals in the future who find themselves in this situation.

Methods: We used the Pubmed and Uptodate databases.

We present the following clinical case.

Male, 36 years old, with a diagnosis of Schizophrenia. Several admissions to the Acute Unit over the years, requiring treatment with ECT. Maintenance treatment with Olanzapine, with which he maintained some delirious ideation and tendency to isolation. He was admitted again in 2023 due to a destabilization of his pathology, presenting delusions of harm, persecution, self-referentiality, auditory hallucinations, imperative phonemes, etc. with important affective and behavioral repercussions. Several pharmacological treatments were tried (Olanzapine, risperidone, aripiprazole), finally the patient showed some improvement with Lurasidone although his functionality was still impaired.

It was decided to start treatment with Clozapine, to minimize the psychotic symptoms, after a hemogram study, which was normal.

Results: During the weekly follow-up of the treatment, a decrease in lymphocytes was observed, with normal neutrophils. The treatment was proving to be ineffective, so it was decided to continue in this line.

Seven months after starting the treatment, the patient suffered a catarrhal process, and once resolved, we observed in addition of the lymphopenia, anaemia and grade 2 neutropenia in the hemogram. Succeeding a consult with hematology specialist we decided to stop the treatment.

The week following the suspension of the treatment, the hemogram normalizes, but the psychotic symptomatology worsens (inability to relate to others, thought blocks, etc.). Taking into account that the blood alterations occured after a cold, and the mental deterioration that the patient presented, it is agreed with the family and the patient to restart the treatment. Wich resulted in improvement of the psychotic symptoms but a new leukopenia due to a slight lymphopenia is observed again.

Conclusions: The average time described for the resolution of severe neutropenia is 12 days. In our case, the hemogram started to improve by the fifth day following the suspensión of the treatment. As it is an infrequent side effect, we do not have studies on the effects of lymphopenia secondary to Clozapine.