Introduction: Akathisia is a relatively common adverse effect of antipsychotics although some second-generation antipsychotics are known to have a lower liability for the condition. The core feature of akathisia is mental unease characterized by a sense of agitation, usually accompanied by motor restlessness, which can cause patients to pace up and down and be unable to stay seated for more than a short time. An association between this discomfiting subjective experience and suicidal ideation has been postulated but remains uncertain.
Objectives: Our aim is to perform a non-systematic review of the literature regarding the current understanding of antipsychotic-induced akathisia and its management.
Methods: A semi-structured review was conducted on Pubmed concerning the relationship between akathisia and antipsychotics.
Results: All antipsychotics drugs can cause akathisia. The management of antipsychotic-induced akathisia should include a dose reduction of the antipsychotic treatment or a switch to quetiapine or olanzapine. If ineffective, a trial with propranolol may be useful as well as the addition of a 5-HT_{2A} antagonist like mirtazapine or mianserine. At last the inclusion of a benzodiazepine may be helpful albeit the risk of dependence and anticholinergics mainly when other extrapyramidal symptoms are present.
Conclusions: High-dose antipsychotic medication, antipsychotic polypharmacy and rapid increase in antipsychotic dosage should be avoided to prevent akathisia. There is limited evidence for any pharmacological treatment for akathisia such as switching to an antipsychotic medication with a lower liability for the condition, or adding a beta-adrenergic blocker, a 5-HT_{2A} antagonist or an anticholinergic agent although some patients may benefit from such interventions.

Keywords: Akathisia; Antipsychotics; extrapyramidal; Anxiety

EPP1054

Quincke-edema induced by chlorpromazine: About two cases.

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doi: 10.1192/j.eurpsy.2021.1288

Introduction: Quincke-edema has been specifically associated with using certain drugs including chlorpromazine as detailed through two clinical cases.
Objectives: Illustration of two clinical cases about angioedema induced by Chlorpromazine.
Methods: We reviewed clinical data from two patients who committed a suicide attempt and then transferred to the psychiatry department after their somatic stabilization: the first was 27-year-old followed in psychiatry since childhood for intellectual deficiency and admitted to the emergency department for the suicide attempt by taking 14 tablets of chlorpromazine 100 mg and the second was a 20-year-old patient, admitted to the emergency department for suicide attempt by Raticid.
Results: The first patient presented a delusional persecution-themed syndrome with auditory hallucinations. Therefore, he was initially put on injectable treatment with Haloperidol 15mg and Diazepam 30mg then oral relay after 48h by Risperidone 4 mg and Chlorpromazine 200 mg. On the fourthday of his hospitalization, he presented a Quincke edema without laryngeal impairment. We stopped chlorpromazine and eliminated the other causes of this edema, resulting in a gradual regression of symptomatology. The second patient was put on chlorpromazine. On the second day, the patient presented a Quincke edema without laryngeal impairment. Somatic examination and biological exploration did not reveal any abnormalities. We stopped chlorpromazine and put the patient on Dexamethasone 3 days in a row resulting in a good outcome.
Conclusions: These two cases identified a Quincke-edema reaction associated with the use of Chlorpromazine, this complication can lead to life-threatening manifestations and warrants greater awareness of the potential for recurrence.

Keywords: chlorpromazine; clinical case; Pharmacology; Quincke-edema

EPP1055

Clozapine-associated eosinophilia with multiple systemic involvement - case report and review of literature

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doi: 10.1192/j.eurpsy.2021.1289

Introduction: Due to its mood-stabilizing properties, clozapine is known for reducing symptom severity in manic episodes of treatment-resistant bipolar disorder as well as in treatment-resistant schizophrenia. However, its use may be hindered by potential adverse effects, including hematologic ones, such as non-dose-dependent eosinophilia. The mechanism of the underlying process probably involves a type-I hypersensitivity reaction, which can manifest as either transient asymptomatic eosinophilia or as eosinophilia with multiorgan dysfunction.
Objectives: We present the case of a patient diagnosed with manic episode of schizoaffective disorder who developed eosinophilia, with severe systemic manifestations, in response to clozapine therapy. A review of literature will be conducted in order to provide further insight into the phenomenon.
Methods: Case report and literature review.
Results: The incidence of eosinophilia reported in literature ranges between 0.2% and 62%, with its appearance about three weeks after clozapine initiation. Although clozapine is an antipsychotic that normally requires frequent monitoring due to the potential side effect of agranulocytosis, we would like to place emphasis on the possible risk of eosinophilia, in connection with potential fatal complications. As described in this report, eosinophilia could long remain unrecognized due to subsequent multiorgan involvement, including lymphadenopathy, leukocytosis, lymphopenia, anemia, liver enzyme elevations, as well as pleural effusion, all of which were described in our patient.
Conclusions: Clozapine-associated eosinophilia may be used as an early marker of possible clozapine-induced systemic complications