

Introduction: Haloperidol is a high-potency first generation antipsychotic and one of the most frequently used antipsychotic medications. It is a potent central antagonist of type 2 dopamine receptors, with low alpha 1 adrenergic activity and has no antihistamine or anti-cholinergic activity. It is a widely used drug with proven efficacy. Angioedema is a very rare side effect, occurring in <1% of cases.

Objectives: Case report and reflection on its etiology

Methods: A Pubmed search was performed with the MeSH terms “haloperidol” and “Anaphylactic reactions”. Relevant articles obtained from the respective bibliographic references were also consulted.

Results: The following case describes the development of angioedema in a patient with an acute confusional syndrome on the second haloperidol IM administration for symptomatic control of agitation. Angioedema has been reported as an adverse effect of various antipsychotics such as clozapine, risperidone, ziprasidone and chlorpromazine, however, resulting from haloperidol administration is rare.

Conclusions: In long-term formulations sensitization testing is especially important but a single prior administration is not sufficient, a second controlled administration is essential to avoid this kind of fatal reactions.

Keywords: Angioedema; Anaphylactic reaction; Haloperidol

EPP1050

Title: Risk factors of prolonged corrected QT interval among patients with mental disorders

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doi: 10.1192/j.eurpsy.2021.1285

Introduction: There is an increased rate of sudden cardiac death in mental health patients. Studies provide consistent evidence that prolonged QT interval is associated with higher risk of all-cause and cardiovascular mortality.

Objectives: This study aimed to assess the prevalence of prolonged QTc interval (corrected QT>450 milliseconds) and to determine the possible factors in hospitalized psychiatric patients.

Methods: We reviewed records of all mental health inpatient admissions to the psychiatry “C” department at Hedi Chaker university hospital in Sfax, between 1 february and 30 april 2019. Electrocardiogram (ECG) availability was noted and QTc interval was manually measured. Sociodemographic, clinical, biological and therapeutic data were collected.

Results: Of 68 mental health inpatient admissions, 59 (86.6 %) presentations had an ECG. A total of seven (11.8 %) had a prolonged QTc interval. These seven patients were treated with typical antipsychotics. Of the 7 patients with a prolonged QTc, 4 patients (57.1%) suffered from schizophrenia. QTc prolongation was significantly correlated with the presence of a recent physical trauma ($p = 0.021$), dietary restriction ($p = 0.026$), and taking at least two antipsychotics ($p = 0.008$). Moreover, this prolongation of QTc was linked to a longer duration of disease and an older age, without significant associations.

Conclusions: Our study supports an association between a prolonged QTc interval and clinical situations at risk and antipsychotic polypharmacy. However, a larger study with routine ECG screening is required to better assess the significance of this problem.

Keywords: Mental disorders; Antipsychotic drugs; Risk factors; Prolonged qt interval

EPP1051

Oxcarbazepine-induced hyponatremia: A case report

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doi: 10.1192/j.eurpsy.2021.1286

Introduction: Oxcarbazepine (OXC) is an antiepileptic drug widely used in the treatment of bipolar disorder (BD), specially when there are side effects with other mood stabilizers. Nevertheless, it isn't innocuous of adverse effects and its consequences can even endanger the patient's life.

Objectives: Brief review of the literature on OXC-induced hyponatremia and exposure of a case report.

Methods: Review of the literature through research in the PubMed database, using the following keywords: “oxcarbazepine”, “hyponatremia” and “adverse effects”.

Results: Although most of the patients are asymptomatic, hyponatremia is one of the most important side effects of OXC. About 29.9% of the patients develop hyponatremia, but only 2.5-3% of psychiatric patients develop severe hyponatremia. The risk of hyponatremia is higher during the first three months of treatment. Severe and/or symptomatic hyponatremia has important clinical implications and may be associated with neurological damage, including seizures, brain stem herniation and death. A 44-year-old woman diagnosed with BD started OXC due to drug intoxications with other mood stabilizers. Six days after initiating treatment, she presented persistent vomiting and severe hyponatremia was detected in blood tests. OXC was suspended with symptomatic resolution.

Conclusions: Healthcare professionals should be alert to symptoms that may arise in patients under OXC. Periodic evaluations of serum sodium levels should be carried out. Cases of severe and/or symptomatic hyponatremia should be rapidly identified and treated in order to reduce the risk of developing brain injury and death.

Keywords: Oxcarbazepine; Hyponatremia; adverse effects

EPP1053

How to manage antipsychotic-induced akathisia

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doi: 10.1192/j.eurpsy.2021.1287

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 fourth day 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