(lithium serum level 1,7 mmol/L). Computed tomography scan of the brain was negative for acute injuries. The electroencephalogram showed triphasic waves (1-1,5 Hz). Encephalopathy secondary to lithium intoxication was diagnosed (probably in the context of acute kidney injury precipitated by hypovolaemia – diarrhoea). Lithium was stopped and intravenous isotonic fluids were given. After 1 week, her myoclonus resolved and over the following week the other signs resolved as well. The patient was later discharged to her daughter's home, with follow-up neurology and psychiatry visits.

Conclusions: Both reversible and irreversible neurotoxicity related to lithium have been reported, specially occurring alongside chronic intoxication. If not addressed, impaired consciousness can lead to coma and death. A high clinical suspicion is needed for prompt diagnosis and treatment (intravenous fluids and sometimes haemodialysis are warranted).

Disclosure of Interest: None Declared

EPV0834

Hepatotoxicity of Clozapine : Case report and brief Review

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Introduction: Clozapine is an effective Atypical antipsychotic used in the treatment of resistant schizophrenia .However it can induce liver dysfunction from a simple transient asymptomatic cytolisis (30 to50 %) toa serious fulminant liver failure (0.001 %).

Objectives: To show the heptotoxicity potential of Clozapine and adress the importance of monitoring the liver function tests in clozapine titration to prevent sever conditions

Methods: A case report of a fifty-year old Tunisian male patient diagnosed with resistant schizophrenia who developed a hepatotoxicity under a low dose of clozapine within five days of treatment .

Results: Mr F is a 50 year old patient diagnosed with schizophrenia in 2018 . He had received various antypical and typical antipsychotic treatments including (Haloperidol , Risperidone , Amisulpride , Olanzapine) at effective doses and minimal periods of six weeks . He had no history of systemic diseases or substance use disorder . He smokes 10 cigarettes a day . He had a history of hepatotoxicity on olanzapine. These medications have failed to resolve the persecutory delusion and auditory hallucinations , and the trial of clozapine was institued . Baseline examination and laboratory tests were normal . The previous antipsychotic medication was not continued and a dose of 25 mg clozapine was administred . A marking drowsiness was present in the fisrt days , so we decided to keep the same dose . Five days later , he had high levels of Liver function test (LFT) : Elevated aspartate (5 times above normal) and alanine aminotransferase levels (4 times above normal) , white blood cell count and bilirubine levels were normal . He had no fever or jaundice . The abdominal examination showed a mild sensiblity in the right upper quadrant . Clozapine was immediatly discontinuated . 24 hours later LFT continued to escalate to 5 times greater then normal . Then it decreased continueosly

Conclusions: Clozapine has a potential of hepatotoxicity even at lower dose . Screening liver function tests must be integrated in survey recommendations of clozapine treatment . Further researches must be conducted to understand the mechanism of this side effect in order to avoid sever conditions .

Disclosure of Interest: None Declared

EPV0835

Neutropenia induced by several second-generation antipsychotics :A case report

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Introduction: Antipsychotic medications remain the mainstay of the treatment of various psychiatric disorders, particularly schizophrenia. However, this therapeutic class can induce a range of side effects. Although the treatment with second generation antipsychotics includes a lower risk for extrapyramidal symptoms as compared to first generation antipsychotics, there are numerous adverse events that can result from atypical antipsychotics. Since the introduction of clozapine, there has been increased awareness regarding antipsychotic-induced hematological side effects.

Objectives: The objective of this case report is to highlight the importance of the management of antipsychotic-induced neutropenia.

Methods: We report a patient with history of schizophrenia who developed neutropenia induced by Haloperidol, Chlorpromazine, Olanzapine, Amisulpride and Aripiprazole.

Results: We present a case of a 43-year-old male patient with a history of schizophrenia, admitted in our department for the management of a state of agitation in the context of a relapse of his condition. On admission, the patient experienced psychotic symptoms, including delusions and auditory hallucinations, in addition to negative symptoms, such as affective flattening, alogia, avolition and asociality. He was then started on 12 mg of Haloperidol and 200 mg of Chlorpromazine with a white blood cells count (WBC) of 5.98 x 10^9/L and absolute neutrophil count (ANC) of 2.52 x 10^9/L (WBC reference range: 4.0-10.0 x 10^9 /L; ANC reference range: 1.5-7.0 x 10^9 /L). The patient did not report adverse events on this medication.

15 days into hospitalization, a mild neutropenia was detected (WBC=3.92 x 10^9 /L and ANC=1.01 x 10^9 /L), leading to a discontinuation of the antipsychotic treatment. No signs of infection were found. After one month, the patient had a normal WBC and ANC. Aripiprazole was discussed as a first alternative and was begun at 5 mg/day and then at 10 mg/day. After one week of treatment with Aripiprazole, the patient's WBC was normal, but the ANC decreased again leading to a moderate neutropenia (ANC=0.91x 10^9 /L). The antipsychotic treatment was once again discontinued and the hematological evaluation found no other

identifiable cause. Afterwards, neither Olanzapine nor Amisulpride showed significant response to this adverse effect. Finally, the administration of Risperidone led to a positive outcome on the WBC and the ANC.
Conclusions: Awareness regarding the hematological side effects of antipsychotics should increase and clinical management of this type of adverse event should be a subject of interest among psychiatrists.

Disclosure of Interest: None Declared

EPV0836

Drug Induced Bullous Lesion Caused By Valproic Acid in Bipolar Affective Disorder:A Case Report

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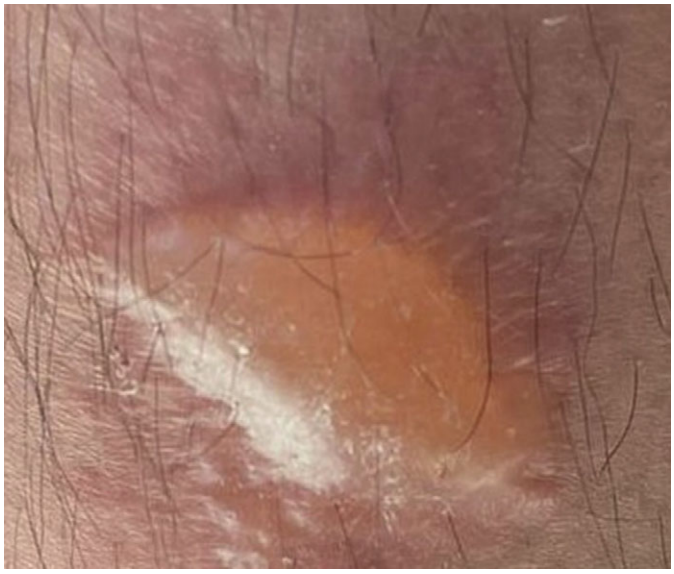
Introduction: Bipolar Affective Disorder(BAPD) is characterized by variations in mood from elation and/or irritability to depression. Valproic acid(VPA) is indicated for the treatment of acute manic episodes in BPAD.The use of VPA can be limited by either loss or lack of efficacy or by adverse drug reactions. Stevens-Johnson syndrome(SJS) toxic epidermal necrolysis(TEN) are the rare but fatal cutaneous adverse drug reactions for VPA.
Objectives: We wanted to draw attention that drug induced bullous lesions which has been seen rarely in the literature caused by valproic acid in bipolar disorder.
Methods: We examined the side effects of valproic acid in one of our patients with bipolar affective disorder using our observations and laboratory tests.
Results: A 41 year old man was admitted to the our hospital with complaints of decreased amount of sleep, increased amount of speech, skepticism,irritability and dysphoric mood.The patient who was followed up with a diagnosis of BPAD for about 10 months,attempted suicide by taking lithium 2 days before his hospitalization.Considering that it was a mixed episode and the prophylactic effect of lithium was insufficient,VPA 1000 mg/day and Risperidone 2 mg/day treatment were started.Risperidone was increased to 4 mg/day because psychotic symptoms persisted.Valproic acid dose was increased to 1000 to 1500mg/day after the Valproic acid blood level reached 55.28 in the follow-ups.After 5 days 2 bullous lesions developed on the lower extremity of the patient.Routine laboratory investigations were within normal limits.When we consult the patient with the dermatologist;the dermatologist recommended that the lesion be fixed drug eruption and that valproic acid should be discontinued if possible.It was thought that the lesions of the patient who did not have dermatological disorders and did not describe insect bites,might be due to valproic acid. In addition to all these,the patient's mother had pemphigus vulgaris.The patient's valproic acid drug was discontinued and lithium was started.Risperidone treatment was continued.In the follow-ups,the patient's bullous lesions regressed and no new lesion formation was observed.

Image:



Conclusions: The differential diagnosis of bullous lesions at first may appear overwhelming.In this case traumatic bulla,Pemphigus vulgaris,drug induced bulla,Fixed drug eruption,Steven Johnson Syndrome were among our prediagnosis.Cutaneous drug eruptions associated with VPA can range from maculopapular eruption to severe Stevens-Johnson syndrome or toxic epidermal necrolysis. We were worried that the patient had SJS, but it remained only bullous lesions.We could not biopsy the patient lesions to understand the underlying cause but development of bullous lesions with the initiation of valproate and subsequent remission of the lesions with the discontinuation of the drug and subsequent course clearly suggests a causal relation between valproate and skin lesions.

Disclosure of Interest: None Declared

EPV0837

Persistent hiccup as an adverse effect of amisulpride in a patient with first episode of psychosis.

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Introduction: A 28-year old male patient was admitted involuntarily to the 4th PICU of the Mental Health Hospital of Thessaloniki, due to severe psychotic symptoms and disorganised behaviour. Upon mental health examination the symptoms included auditory hallucinations, tangible speech, delusional ideas of somatic and persecutory type and significant neglect of his personal hygiene. The onset of his psychotic illness was 3 years prior, with two hospitalizations in the UK, and several unsuccessful attempts of outpatient monitoring.