(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).

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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 .

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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