Objectives: To describe a case of severe NMS and review the key elements of differential diagnosis and treatment options.
Methods: A case report and a non-systematic review of the literature.
Results: A 54-year-old male was admitted to the hospital after being found unconscious on the street during a hot August day, with apparent cranioencephalic trauma and a core temperature of 41°C. The patient had a known history of chronic schizophrenia, managed with risperidone (6 mg/day) and quetiapine (800 mg/day), with no recent medication changes. Initial examination in the Emergency Department revealed tachycardia, tachypnea, normotension, diaphoresis and confusion. Laboratory results showed mild leukocytosis with neutrophilia, hyperkalemia, hypernatremia, and elevated acute phase reactants, including a creatine kinase (CK) level exceeding 3000 IU/L. Lactic acidosis and impaired renal function (creatinine 1.9 mg/dL) were also noted. Infectious causes were ruled out, and neuroimaging did not reveal any acute findings to account for the symptoms. Due to persistent decreased consciousness, the patient required intubation and supportive care in the Intensive Care Unit (ICU).
The patient exhibited persistent hyperthermia that was unresponsive to antipyretics and physical cooling measures. Upon suspicion of NMS, antipsychotic medications were discontinued, and treatment with a dopamine agonist and dantrolene was initiated. Muscle rigidity, which developed later, further supported the NMS diagnosis. Electroencephalography (EEG) findings were consistent with NMS. Given the patient's slow recovery, electroconvulsive therapy (ECT) was started but discontinued after two sessions due to ICU-related infectious complications.
After 19 days of hospitalization and treatment, the patient showed significant clinical improvement, allowing for extubation and discontinuation of intensive care interventions.
Conclusions: This case emphasizes the importance of early recognition and treatment of Neuroleptic Malignant Syndrome to avoid complications and mortality. Differentiating NMS from classical heat stroke can be challenging, with muscle rigidity serving as a critical diagnostic feature.

Disclosure of Interest: None Declared

EPV1582

Edema associated with Haloperidol: A Rare Observation

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Introduction: Haloperidol (Haldol®) is first-generation antipsychotic that still have a place in the treatment of schizophrenia. However, this molecule is associated with numerous side effects, particularly extrapyramidal symptoms. Edema has been rarely described with haloperidol, and may have an immunoallergic or pharmacodynamic mechanism.
Objectives: Our aim is to study a rare case of facial and limb edema attributed to haloperidol.
Methods: We report the case of a 22-year-old female patient with schizophrenia who developed facial and limb edema after taking haloperidol.
Results: We report the case of a 22-year-old woman. She was admitted to our psychiatric department "A" as an involuntary inpatient after she threatened to stab her father. She was diagnosed with schizophrenia. The patient has no past medical history. The somatic examination and the admission report were correct.
Given the need for a slow-release neuroleptic, the patient was treated with haloperidol in gradually increasing doses up to 30 mg per day. Six weeks after starting treatment, the patient developed progressive edema of the face, eyelids and all 4 limbs, with a marked increase in weight and body mass index from 31 to 36.6.
The patient was examined by internists. An etiologic investigation of this edema was initiated, excluding renal origin (strictly normal renal work-up and negative proteinuria), cardiac origin (in view of a negative pro-brain natriuretic peptide, D-dimer and troponins and a normal cardiac echography), hepatic (in the presence of a strictly normal work-up including: protidemia, albuminemia, transaminases, PAL, GGT, bilirubin and prothrombin levels) and endocrine (a normal thyroid work-up) origin.
The edema progressively worsened on haloperidol over a period of 10 days. A drug-induced origin was suspected. We contacted the regional pharmacovigilance center in Sfax, which incriminated haloperidol and recommended its immediate discontinuation.
After discontinuing the drug, the edema regressed progressively until it disappeared completely after 15 days, confirming that haloperidol was the drug responsible and contraindicating its further use.
The responsibility of haloperidol in the genesis of the edema was retained with a plausible score (C2S2I2B3). The scores were calculated according to the French Bégaud method.
Conclusions: Edema is not a common side effect of typical antipsychotics, especially haloperidol. However, it is important to emphasize that this effect, although rare, can occur. Clinicians are advised to be aware of edema in all patients taking first or second antipsychotics.

Disclosure of Interest: None Declared

EPV1583

Chlorpromazine induced acute cytolytic hepatitis: Case report and a literature review

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Introduction: Chlorpromazine is historically the first antipsychotic drug. It has played a decisive role in the development of neuropsychopharmacology. However, it can induce adverse effects, sometimes fatal.
Objectives: To study the relationship between acute cytolytic hepatitis and treatment with chlorpromazine.
Methods: We report the case of a patient treated for schizophrenia who developed an acute cytolytic hepatitis after taking chlorpromazine.