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КНИГА НА АПСТРАКТИ

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Chronic lithium toxicity

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Objective: Lithium toxicity can be acute or chronic and the effects may be disabling or life-threatening. We present a case with lithium toxicity due to chronic treatment.

A 63-year-old man was brought to the University Clinic for Toxicology in Skopje with a 2-day history of confusion, slurred speech, nausea, vomiting, loss of appetite, and weakness. His medical history included bipolar disorder, which had been diagnosed 17 years previously, and since then he has been on lithium therapy 900mg orally daily. The patient had not had regular check-ups for several years. His oral intake was markedly reduced, for the last five days. On admission, he was severely dehydrated. Vital signs included a blood pressure of 110/50 mm Hg a pulse rate of 49 beats/min, and oxygen saturation of 97%. Investigations revealed serum lithium level 3.43 (normal 0.5–1.5 mmol/L) suggesting lithium toxicity, creatinine level 168 umol/L, urea 15 mmol/L alkaline phosphatase 182 U/L calcium 2.34 mmol/L. A 12-lead electrocardiogram showed normal sinus rhythm, precordial T-wave inversions, and a prolonged QTc interval of 533 ms. After one treatment of hemodialysis lasting two and half hours, his lithium level dropped to 2,04 mmol/L and creatinine to126 µmol/L. No intracranial abnormality was seen on a computed tomography scan of the head. Further investigation revealed parathyroid hormone 170 pg/ml, ionized calcium 1.43 mmol/L. Treatment management included volume replacement for dehydration. Abdominal computed tomography was with orderly finding. All investigations were at the normal therapeutic range before discharge and sent to the psychiatrist on the eleventh day.
Conclusion: Patients on long-term lithium therapy with no regular check-ups are at high risk of developing toxicity. The toxicity was due to intravascular volume depletion, and impaired lithium excretion. The patients should be monitored frequently for all potential endocrine, renal, and neurological disorders.
Key words: lithium, toxicity, parathyroid hormone

Opioid overdose or other somatic comorbidity - fatal case

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The aim of this case presentation is to emphasize the diagnostic challenges that the clinicians encounter when dealing with a comatose patient and the importance of keeping a broad differential diagnostic panel in mind.
Case report: A 47-year-old female patient, was brought by ambulance to the University Clinic for Toxicology in Skopje. On admission, she was comatose (GCS=5), with miotic isochoric pupils, blood pressure was 90/60 mmHg, with oxygen saturation from 85 up to 92%. The obtained data from family indicated that the patient was with opioid use disorder on methadone maintenance therapy. Recently, the patient has consumed large amounts of alcohol . The family's suspicion was that perhaps the new condition was caused by excessive intake of alcohol or methadone or both. In the meantime, the result of alcoholemia showed 67.0 mg/dL (value <100mg/dL-low level) and the toxicological screening in urine sample for tetrahydrocannabinol, opiates, tramadol, amphetamine, 3,4-methyl-enedioxy-methamphetamine, cocaine, benzodiazepines, buprenorphine was negative with mildly elevated methadone values (the patient was on methadone substitute the last 7 years). Second day on physical examination a brisk response to deep tendon reflexes of the left side of the body with apparent right hemiplegia was noted. Computed tomography of the brain was performed immediately and showed an ischemic stroke with a compressive effect on the left lateral chamber. Although it was immediately started with an aggressive treatment, after 11 days the condition of the patient deteriorated and resulted in death.
Conclusion: The notable opioid prevalence,mandates that physicians maintain a high index of suspicion when dealing with a comatose patient, especially if the patient has any known history of opioid abuse. Healthcare professionals should be aware that a comatose state in a patient could be caused by either non-toxicological trigger or by toxic causes.
Key words: alcohol, methadone, ischemic stroke, coma

Severe systemic toxicity after intravenous administration of metamizole and ceftriaxone in a splenectomised patient-case report

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Drug-induced toxicity can have a mild to severe clinical presentation as a life-threatening condition. We presented a case with a general vasculitis and severe multi-organ failure in a splenectomised middle-aged woman, which developed after ceftriaxone and metamizole parenteral administration. A middle-aged woman was treated with IV metamizole and ceftriaxone for a fever and soar throat in a local hospital. She had a post traumatic splenectomy 5 years ago. After metamizole, during ceftriaxone administration she felt burning in her face with maculo-papulose rash which started to conflate, spread to whole body and intensively darkened. She was transferred to the University Toxicology Clinic with a hypotension, hypoxemia, generalized necrotic vasculitis with predominant facial distribution. There was increased values for CRP (250 mg/l), WBC (27x10^9/l) and LDH (1867 U/l) during hospitalisation. She also presented anaemia (Er 2.6x10^12/l, Hgb 88 g/l, Hct 0.24), polyserositis-ascites, pleural effusion and mild pericarditis (high sensitive troponin 107 ng/l), acute pancreatitis (amylase 1048 U/l, lipase 881 U/l), hepatomegaly, acute kidney injury (BUN 36.5 mmol/l, creatinine 528 µmol/l, oliguria), disseminated intravascular coagulation (Plt 23x10^^{9}/l, DD 7658ng/ml, PT 56 sec, aPTT 120 sec), vitreous haemorrhage of the right eye and rhabdomyolysis, CPK 428U/l. Microbiological findings were negative. Immunoserology showed positive p-ANCA. The acute renal failure, ascites and pleural effusions resolved under methylprednisolone, meropenem, LMWH, haemodialysis and symptomatic therapy, with normalization of laboratory parameters. A skin biopsy finding was inconclusive. After 25 days, rheumatologist recommended mycophenolate mofetil PO. She was asymptomatic with prednisolone and mycophenolate mofetil therapy during following two years and maintained stable after their discontinuation.
Drug-induced toxicity have potential to induce a severe multiorgan failure with life-threatening complications. Splenectomy may be studied as a potentially risk factor for immunomodulated response to drugs and drugs interactions, especially during infections.
Key words: drug-toxicity, vasculitis, p-ANCA, multiorgan failure