Severe cognitive decline following chemotherapy of breast cancer

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Chemotherapy for different types of cancer is often life-saving. On the other hand, the spectrum of side effects may involve the central nervous system (CNS) and lead to severe emotional and cognitive disturbances, a syndrome often referred to as “The Chemo Brain”.

Here we report on a then 43-year-old female patient who first presented to our clinic in 2013 and in whom breast cancer of the right breast had been diagnosed four years earlier. At that time she became a patient of the Comprehensive Cancer Center of the University of Ulm (CCCU) and underwent breast-conserving therapy starting with systemic chemotherapy according to the rules of the GeparQuinto Study. This was followed by breast-conserving operation including axillary lymphonodecctomy. Histological examination of the excised material revealed an invasive ductal mammary carcinoma the exact staging of which will be reported in detail. Convalescence of the patient was protracted, and she continued complaining of difficulties to concentrate and to remember what she had been told or read shortly before. In addition, she complained of frequent mood swings. Therefore, she was referred to our department. Exploring her case history, we learned that she had worked as a manager of a car rental company which she had no longer been able to do after her cancer treatment because of her cognitive deficits. Beside clinical and psychiatric examination, our diagnostic procedures included electroencephalography (EEG), magnetic resonance tomography (MRT) of the brain using gadolinium enhancement, and extensive neuropsychological testing. In detail, components of the Wechsler Adult Intelligence Scale (WAIS), the Ray Auditory Verbal Learning Test (RAVLT), the Multiple Choice Vocabulary (“Mehrfachwahl-Wortschatztest”), the “Regensburger Wortflüssigkeitstest” (Regensburg Word Fluency Test, RWT), and the “Testbatterie zur Aufmerksamkeitsprüfung” (Test Battery for the Assessment of Attention, TAP) were applied.

Whereas the EEG was unremarkable, the MRT performed in 2013 showed brain atrophy with frontotemporal accentuation. As an accessory finding, an arachnoid cyst was detected at the left temporal pole. No metastases were found. Interestingly, we had the opportunity to compare these findings with those of an earlier MRT performed in 2011. In the latter one, the arachnoid cyst was also visible, but there was no pronounced brain atrophy.

These observations raise the question if progressive brain atrophy might be associated with the syndrome of “Chemo Brain”. Neuropsychological examination revealed severe cognitive deficits in this patient – details of which will be reported – apparently due to the chemotherapy she had undergone. Possible mechanisms of action, treatment options, and ethical implications will be discussed.

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A rare presentation of Potassium iodate toxicity

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Keywords: Potassium iodide overdose; resistant hyperkalemia; rhabdomyolysis; Acute kidney disease; Methemoglobinemia; Aspiration pneumonia; Fluid overload secondary to Renal failure; Bilateral vitreous hemorrhage with posterior vitreous detachment

Introduction: Potassium iodate is a compound used for nutritional supplementation in table salt and administered to individuals exposed to radioactive iodine. Toxicity to Potassium iodate is not a common presentation to the emergency room. In this case report, we present a young man who was brought to the ED after deliberate oral ingestion of Potassium iodate, who then had a remarkable clinical course in the hospital.

Case description: A 26-year-old Sri Lankan male was referred to the Emergency department as a case of ST elevation MI from the primary health centre, where he had presented with diarrhoea. In the Emergency, he admitted to having consumed a “handful” of potassium iodate powder, 17 hours prior to presentation to ED, as an act of deliberate self-harm. He then had complains of cramping abdominal pain, loose stools and vomiting. He was also complaining of visual disturbances, in the form of seeing everything with a greisy tinge.

On initial assessment of patient, he was talking, with a patent airway, no evidence of any respiratory distress and had stable haemodynamics and GCS of 14/15. Visual acuity was 6/6 but stated everything appears grey.

ECG showed ST elevation and tall T waves in chest leads, reciprocal changes of ST depression in III, aVF with prolonged PR and QRS interval. Echocardiogram was done which showed no regional wall motion abnormality. Labs showed high potassium (K-7.6meq/L). Other notable results were CPK of 3909U/L, myoglobin more than 5000ng/ml, high sensitive Troponin T - 220ng/L, Troponin I - 1.8ng/L. Leukocytosis of 34500/μL with neutrophilia, creatinine - 226μmol/L, BUN - 6.07mmol/L Sodium - 139meq/L, mixed respiratory and metabolic acidosis with normal anion gap and osmolar gap. Fibreoptic laryngoscopy looking for any features of corrosive damage to upper airway proved normal. Over few hours he developed methaemoglobinaemia.

Treatment was started for hyperkalemia but only had transient response to repeated medical management. Emergency hemodialysis was started to treat the hyperkalaemia. He was later admitted under the intensive care with input from renal team. He received hemodialysis for more than 3 weeks. He was also found to have only light perception in both eyes with bilateral vitreous hemorrhage and posterior vitreous detachment. Patient as of date has started to pass urine. He is off hemodialysis. He still has renal failure and is continuing treatment in the medical floor.

Conclusion: Though renal failure, hyperkalaemia and retinopathy have been described in literature, the presentation and clinical course of this patient were unique in many ways including severe rhabdomyolysis leading to acute tubular necrosis and bilateral vitreous hemorrhage with posterior vitreous detachment.