Dexamethasone Abuse with Fatal CNS Complication

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A 24 yrs old unmarried chronic alcoholic male, compounder by occupation was admitted with multiple patchy discolouration of skin and swelling all over the body for 2 months and sudden onset altered sensorium for one day. History of self medication in the form of intravenous injection of dexamethasone for last 1½ years was also present. History of sexual contact was not available. History of fever, headache, vomiting, seizure, loose motions, bleeding, ear discharge, jaundice, hematemesis / melena, tuberculosis or ATT intake and diabetes mellitus were absent. On examination, he was obese, vitals were stable and smell of alcohol was absent. Cervical hump, moon facies and lower abdominal purple striae were present. Cutaneous examination revealed multiple annular lesion with hyperpigmented margin and centre clearing along with ulcerative whitish lesions present in the oral cavity and plaques all over the genital region and cleft suggestive of candidiasis. Lesions were also present in between toes. In CNS examination patient was unconscious, responding to deep painful stimuli, focal neurologic deficit and signs of meningeal irritation were absent, bilateral pupils were normal in size and reaction and bilateral plantars were non-elicitable. Rest of the examinations were normal. Provisional diagnosis of chronic alcoholism with intravenous dexamethasone abuse with iatrogenic Cushings with tinea corporis with oral and genital candidiasis with intertrigo with encephalopathy cause ?infection, stroke, metabolic was kept. Emergency NCCT head showed multiple hypodense lesion in both cerebral hemisphere suggestive of infarct. He was started on intravenous mannitol, dexamethasone, dextrose along with multivitamins. Tablet ecosprin, fluconazole and cetirizine, clotrimazole and candid B ointment and betadine lotion for local application.

Investigations revealed hemoglobin-10.7 g/dl, total leucocyte count- 4800/mm³, differential leucocytes count-polymorph 72%, lymphocytes25%, eosinophils 3%, and platelet count - 49000/mm³. Blood urea 51 mg/dl, serum creatinine 1.1 mg/dl, serum sodium 146 mEq/l, serum potassium 4.4 mEq/l, random blood sugar 88 mg/dl, total bilirubin 1.3 mg/dl, aspartate transaminase 258 U/l, alanine transaminase 265 U/l, alkaline phosphatase 70 U/l, total protein 5.1 g/dl (serum albumin 2.0 g/dl). Fundoscopy and chest radiograph were normal. ELISA for HIV 1 & 2 – non-reactive. Blood and urine culture – sterile.

Subsequently on 4th day of admission, he developed moderate grade fever and there was no improvement in sensorium. MRI brain followed by lumbar puncture and echocardiography were planned but unfortunately patient expired. MRI brain with contrast done before the demise revealed multiple discrete and confluent rounded lesions infiltrating both cerebral and cerebellar hemispheres with calcification / hemorrhage and faint peripheral rim enhancement with mild to moderate perilesional edema causing effacement of sulci and feature of early hydrocephalus with mild cerebral atrophy.

Fig.1 : MRI brain with contrast showing multiple ring enhancing lesions with perilesional edema, predominantly involving corticomedullary junction and basal ganglion causing effacement of sulci and feature of early hydrocephalus with mild cerebral atrophy.

Dexamethasone is known for its use in critically ill patients but we have not seen earlier any case of dexamethasone abuse. Patients with AIDS and those receiving immunosuppressive therapy for lymphoproliferative disorder are at greatest risk for developing acute toxoplasmosis. Signs and symptoms principally involve the CNS in the form of encephalopathy, meningoencephalitis and mass lesions.1 The infection has a predilection for the grey-white junction, basal ganglia and thalamus. Though the lesions are typically supratentorial, they have been also noted in the cerebellum and brain stem and rarely in intraventricular locations.2 M  usually demonstrates multiple lesions. These findings are not pathognomonic of toxoplasmosis, since 40% of CNS lymphoma are multiple and 50% are ring enhancing. The findings of a single lesion on MRI scan increases the likelihood of primary CNS lymphoma and strengthens the argument for a brain biopsy. A therapeutic trials of anti-Toxoplasma medications is frequently used to assess the diagnosis.3 Presumptive treatment with pyrimethamine plus clindamycin results in quantifiable clinical improvement in >50% of patients by day 3. By day 7, >90% of treated patients show evidence of improvement.4 In contrast if patients fail to respond or have lymphoma clinical signs and symptoms worsen by day 7. In future single photon emission CT ( SPECT ) and PCR for parasite may prove diagnostically beneficial.

Primary CNS lymphoma is rare in general community but affect about 2% of AIDS patients. It is a typical end stage complication of HIV disease and survival after diagnosis is limited to few months only.5 These patients can achieve some palliation by cranial irradiation.

References


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