ORIGINAL RESEARCH PAPER
Volume - 9 | Issue - 9 | September - 2020 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

AN UNUSUAL CASE OF NEUROLEPTIC MALIGNANT SYNDROME WITH AFEBRILE PRESENTATION

General Medicine

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ABSTRACT

Neuroleptic malignant syndrome is a rare but life threatening neurological emergency associated with the sudden withdrawel of Neuroleptic agents & characterized by distinctive clinical syndromes with Mental status changes, Rigidity, Dysautonomia and Fever are the classical components .Incidence of Neuroleptic malignant syndrome is 0.02 to 3 % among patients with sudden withdrawel of Dopaminergic agents .Age is not a risk factor .Men's are more affected than Women. Pathogenesis of NMLS is that exact Cause is unknown,.Dopamine receptor blockade theory is central to most theories of its pathogenesis. Disrupted modulation of sympathetic nervous system is another accepted theory. Typical course of clinical manifestations includes Mental status changes ,Rigidity, Hyperthermia ,Autonomic dysfunction, Fever appearance may be delayed >24hrs leading to diagnostic confusion. Range of illness limits with Agitated delirium with confusion ,Catatonic signs & Profound mutism ,encephalopathy,Stupor and even Coma.In our case the classical component Fever was absent and presented as afebrile illness.

KEYWORDS

afebrile, creatinine kinase, neuroleptic malignant syndrome.

Introduction

Neuroleptic malignant syndrome is a rare but life threatening neurological emergency associated with the sudden withdrawel of Neuroleptic agents & characterized by distinctive clinical syndromes with Mental status changes, Rigidity, Dysautonomia and Fever are the classical components.An abnormal clinical presentation with afebrile illness can also manifest very rarely in some cases.

Case Report

A 69 year-old male was admitted to the hospital in view of decreased response to oral commands, poor oral intake and altered mental status. Patient was apparently normal prior to this.He a known case of Schizophrenia and Bipolar disorder for the past 14 years with history of recurrent hospital admissions.

On reviewing his previous medical records it is noted that he had been on- Lithium (450mg OD), Quetiapine (100mg OD) ,clonazepam (0.5mg OD), Haloperidol (2mg OD) and Divalproex sodium (500MG OD).

All those above mentioned drugs were suddenly withdrawed by the patient for 48 hours by the patient and history of consumption of Haloperidol 6mg out of recommended dosage was present . He is not a known smoker,alcoholic or illicit drug user.There is no history of similar illness in family and he had no history of drug allergy.

Examination

On examination, patient was drowsy and disoriented with GCS score of 10/15, he was in dehydrated status with poor dental and oral hygiene. Patient was normotensive-BP120 /80mmHg with a pulse rate of 96/min, regular in rate and rhythm, normal volume and character, no radio-radio or radio-femoral delay. Respiratory rate was 20 per minute. His CBG was 155mg/dl. There was B/L pitting pedal oedema and pallor present.Bilateral Pupils were equally and reacting to light.On Central nervous system examination with Generalized rigidity Cog-wheel rigidity was present. Bilateral plantar was non response. Other system examination was normal.

Investigations

All routine investigations were done. His T.Bilirubin 1.2 mg/dL (0.3-1.1 mg/dL), D.Bilirubin 0.9 mg/dl(00-0.2 mg/dl), I.Bilirubin 0.3 mg/dl , AST(SGOT) 99 IU/L (<35 IU/L), ALT(SGPT) 106 IU/l (<45 IU/L),Gamma GGT 63 IU/l (<55IU/l) , A/G Ratio was 1.1, Urea 62 mg/dl (17.12-49.2 mg/dl) ,CPK-MB 5000 IU/L (Up to 24IU/L).His S.Calcium s.ammonia ,Alkaline phosphatase and creatinine level were normal.vel wer

Radiology

His chest X-ray showedshowed no significant finding. CT brain was done showed age related atrophic changes .

Diagnostic scoring

NMS score-67/100[consistent with a diagnosis of NMS]

Management

The patient came to hospital with above mentioned complains. Patient was started on C.Dantrolene 25mg BD,T.Bromocriptine 2.5mg od, T.Syndopa 110mg QID, Lorazepam 1 mg IV BD, Amantadine 100mg OD,T.Trihexyphenidyl HCL 2mg, inj.Piracetam 3g in 100ml of NS and other supportive measures were given. Over the course of 2 weeks, patient showed significant improvement, being able to take oral feed, respond to verbal commands, and walk .Repeated CPK-MB was done and was 16 IU/L (Up to 24IU/L).Hence patient is been discharged and advised followup.

Discussion

Neuroleptic malignant syndrome is a rare but life threatening neurological emergency associated with the sudden with drawel of Neuroleptic agents & characterized by distinctive clinical syndromes with Mental status changes, Rigidity, Dysautonomia and Fever are the classical components. An abnormal clinical presentation with afebrile illness can also manifest very rarely in some cases. NMS is most often seen with HIGH POTENCY 1st Generation Neuroleptic agents. Onset is that it Develops during the first 2weeks of neuroleptic theraphy[1]. Risk factors includes High doses ,Recent rapid dose escalation, Switch from one agent to another, Parenteral administration, Not dose dependent phenomenon[2] .Associated risk factors includes Acute catatonia, Extreme agitation ,Neurologic diseases (Schizophrenia),Acute medical illness (Trauma , Surgery & infection) Antiparkinson medication withdrawal. Distinct disorder from Afebrile NMS are Neuroleptic malignant like syndrome, Parkinsonism hyperreflexia syndrome, Acute Malignant syndrome in Parkinson disease, akinesis. Pathogenesis of Neuroleptic malignant is not fully well understood. Dopamine receptor blockade theory is central to most theories of its pathogenesis. Typical course of clinical manifestations Mental status changes, Rigidity, Hyperthermia, Autonomic dysfunction, Agitated delirium with confusion, Catatonic signs&mutism,Profound encephalopathy, Stupor, Coma, Rigidity Generalized, Lead pipe rigidity, Cog-wheel if superimposed tremor present. Hyperthermia in NMS >38 ºc, Fever less consistent with 2nd generation antipsychotics. Autonomic instability includes Diaphoresis, Labile or high blood pressure, Tachycardia. Other motor abnormalities include Tremor, Dystonia, Opisthotonus ,Trismus, Chorea, Sialorrhea,Dysarthria,Dysphagia[3].Laboratory abnormalities Elevated serum CK: More than 1000 IU/L but Can be as high as 100,000 IU/L.Other non specific lab abnormalities: Leukocytosis (10,000 to 40,000),Mild elevation in LDH , Transaminases and ALP, Hypocalcemia, Hypomagnesemia, Hypernatremia, Hyperkalemia, Metabo-lic acidosis,Myoglobinuric

renal failure – rhabdomyolysis, Low serum Iron concentration Complications Rhabdomyolysis, Acute renal failure, Acute respiratory failre(Pulmonary emoblism), Aspirationpneumonia, Seizures, Braindamage, Myocardia, infarction, DIC, Hepatic failure,E.coli fasciitis,Sepsis[4].Management is by reasonable approach is to start with benzodiazepines along with dantrolene in moderate or severe cases, followed by the administration of bromocriptine or amantadine. Stop causative agent.ICU - supportive care aimed to avoid complications[5].

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